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THE MEDICAL CLINICS OF NORTH AMERICA

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CONTRIBUTORS TO THIS NUMBER

FRANK B. BERRY, M. D., Assistant Pathologist, Boston City Hospital.

IDA M. CANNON, Chief of Social Service, Social Service Department, Massachusetts Genera Hospital.

STANLEY COBB, M. D., Assistant Professor of Neuropathology, Harvard Medical School; Assistant Neurologist, Massachusetts General Hospital.

M. J. ENGLISH, M. D., Assistant in Medicine, Harvard Medical School; Assistant Visiting Physician, Boston City Hospital.

JOSEPH I. GROVER, M. D., Assistant in Pediatrics, Harvard Medical School; Junior Assisting Physician of the Children's Hospital.

LEWIS WEBB HILL, M. D., Junior Assistant Visiting Physician, Children's Hospital; Alumni Assistant in Pediatrics, Harvard Medical School, Boston.

ALBERT A. HORNOR, M. D., Second Assistant Visiting Physician, Boston City Hospital.

MAYNARD LADD, M. D., Instructor in Pediatrics, Harvard Medical School; Physician-in-Chief, Children's Department of the Boston Dispensary; Consulting Physician to the Boston Children's Hospital.

EDWIN A. LOCKE, M. D., Assistant Professor of Medicine, Harvard Medical School; Visiting Physician, Boston City Hospital.

JOHN LOVETT MORSE, M. D., Professor of Pediatrics, Harvard Medical School; Visiting Physician, Infant's Hospital and Children's Hospital, Boston.

H. ARCHIBALD NISSEN, M. D., Alumni Assistant in Medicine, Harvard Medical School; Second Assistant Visiting Physician, Boston City Hospital.

EDWARD H. NICHOLS, M. D., Professor of Clinical Surgery, Harvard Medical School; Attending Surgeon, Boston City Hospital.

W. RICHARD OHLER, M. D., Assistant in Medicine, Harvard Medical School; Second Assistant Visiting Physician in Charge of Clinical Pathological Laboratory, Boston City Hospital.

KARLTON G. PERCY, M. D., Assistant Visiting Physician to the Children's Hospital, Boston; Instructor in Pediatrics, Harvard Medical School.

WILLIAM D. REID, M. D., Assistant Visiting Physician to Out-patient Department, Massachusetts General Hospital; Second Assistant Visiting Physician, Boston City Hospital.

WILLIAM H. ROBEY, JR., M. D., Assistant Professor of Medicine, Harvard Medical School; Visiting Physician to Boston City Hospital.

PHILIP H. SYLVESTER, M. D., Instructor in Pediatrics, Harvard Medical School; Assistant Visiting Physician to the Children's Hospital.

FRANKLIN W. WHITE, M. D., Instructor in Medicine, Harvard Medical School; Visiting Physician, Boston City Hospital.

PAUL D. WHITE, M. D., Fellow in Medicine, Harvard University; House Physician and in Charge of Cardiac Clinic, Massachusetts General Hospital.

EDWIN T. WYMAN, M. D., Assistant in Pediatrics, Harvard Medical School; Junior Assistant Visiting Physician, Children's Hospital; Visiting Physician, Boston Floating Hospital.

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THE MEDICAL CLINICS
OF
NORTH AMERICA

VOLUME 4 NUMBER 2

CONTRIBUTION BY IDA M. CANNON

CHIEF OF SOCIAL SERVICE DEPARTMENT, MASSACHUSETTS GENERAL HOSPITAL

A MEDICAL-SOCIAL CLINIC

PROGRESS in the organization of scientific medicine has been made through differentiation of function, based on special knowledge and special skill. The institutionalizing of the practice of medicine has made this special knowledge and special skill available to a large number of persons. In this process of organization, however, medicine lost what is well recognized as an essential element in successful medical practice—a personal knowledge and a sympathetic understanding of the patient and of the social conditions of his life.

The general trend of medical treatment during the past twenty years has been distinctively away from pills and powders. Hygienic living, with carefully regulated diet, rest, exercise, or work, is often more effective than specific drugs. The doctor must diagnose and prescribe, but successful treatment in a great multitude of cases requires that the patient become a partner in the treatment. The diabetic patient cannot be successfully treated unless his co-operation is secured in maintaining rigidly a restricted diet. The organic heart case cannot be controlled by drugs alone; the patient must learn to live within his limitations. The patient with scoliosis must himself carry on the prescribed exercises persistently. The psychoneurotic patient will be greatly helped if he is furnished with some interesting

occupation. These principles are well recognized by physicians in their private practice.

Now, the function of the social worker in the hospital is to obtain for the physician such information concerning these personal and community factors as is essential to prognosis and treatment, and, by her wisdom and skill in adjusting environment, in influencing, guiding, and controlling the patient's conduct, to make that treatment effective.

The following analysis of the combined medical-social diagnosis of a ten-year-old Syrian boy, discharged from the hospital "Relieved," illustrates the interrelation of the function of the doctor and the social worker:

- Medical-social diagnosis
  - Personal
    - 1. Physical
      - Acute.
      - Endocarditis.
    - 2. Mental
      - Normal.
      - Intelligent.
  - Environmental
    - 3. Physical
      - Poor home, third floor; crowded district.
      - Inadequate food.
      - Second floor school rooms; the crowded street only playground.
    - 4. Mental
      - Half-orphan.
      - Mother incompetent.
      - Lack of discipline.

The physician determines factors numbered 1 and 2. The social worker may contribute to No. 2 by bringing to the doctor facts about the patient's behavior in border-line mental cases. The doctor on the staff of a big dispensary clinic has not time or opportunity himself to secure factors numbered 3 and 4. They must be determined by a knowledge of the patient, his home, his work, his companions, his interests, and it is here that the social worker becomes necessary. A correct prognosis or an intelligent plan of treatment often cannot be reached without a knowledge of all these personal and environmental factors—the patient's physical and mental condition, his physical environment, and the mental atmosphere in which he lives. In

the case used for illustration the prognosis, as well as the treatment followed, were materially modified by the report of the social worker concerning these conditions. It was found necessary, in the light of this additional information, to place the patient in a foster home, where he was kept for several months, until his heart was in good condition, and until habits of living had been established that might prevent a recurrence; while supervision, both medical and social, was necessary for several years. The social worker took the responsibility of seeing that the patient came regularly, at appointed times, to the dispensary.

The following cases are presented as illustrating the bearing of the social facts on prognosis, and the interrelation of social and medical treatment:

Case A.—M. M., a man of forty-seven; native of Ireland; freight handler, was admitted to the male clinic on August 12, 1914, following hemorrhage of the lungs.

Family history taken by the doctor at the clinic showed nothing significant. Patient was said to have two brothers and two sisters in good health. His wife was "well," and he had five "healthy" children.

The patient's habits were essentially normal; his past medical history negative, except that he had "coughed for years," and "more in the morning." There was a history of a loss of 10 pounds in weight during the previous three months; occasional night-sweats; some vomiting of greenish substance. On August 11th, the day before admission to the clinic, he had raised a large quantity of blood, which had led him to seek medical aid at the hospital.

A diagnosis of pulmonary tuberculosis, moderately advanced, was made, and the patient was referred to the Social Service Department to arrange for his admission to a sanatorium.

The patient was loath to give up his work because of his feeling of responsibility for his family; but, after a home visit, he was persuaded by the social worker to accept interim care in a local hospital, pending admission to the state sanatorium.

The social history disclosed the following facts:

The family consisted of a wife, age forty-one; a daughter, age eight; a daughter, age six; twin boys, age four, and a baby girl of seventeen months.

Patient had lived on a farm in Ireland until the age of twenty-five. He was said to have had rather unusual school advantages, in view of his social status and the community in which he lived. He emigrated to this country at twenty-five. From that time he had done various kinds of unskilled labor; had worked steadily, and had usually done rather arduous and poorly paid work. At this time he was receiving $13.80 a week. There were no savings. The income was obviously not sufficient to provide the proper amount of nourishing food. The family were living in a relatively comfortable house, which was scrupulously clean, and the wife, an American by birth, was, apparently, a capable woman, who used what she had to the best advantage.

Patient had no appreciation of the necessity of well-ventilated sleeping rooms. Dental hygiene he entirely neglected; he was otherwise clean about his person.

None of the relatives were in a position to assume financial responsibility for the family.

The mother gave a history of one of the twins having been to a local dispensary six months earlier. Inquiry at the dispensary disclosed the fact that at that time a diagnosis of pulmonary tuberculosis had been made; but no treatment had been given, and the case had not been followed.

The wife and children were brought to the clinic for examination. The medical report was as follows:

Mother (forty-one years), quiescent tuberculosis of lungs.
Katharine (eight years), tubercular glands.
Margaret (six years), tubercular glands.
James (four years), tuberculosis.
Fred (four years), tuberculosis.
Mary (seventeen months), tubercular glands.

This report was a great shock to the mother, who already had been much disturbed by the knowledge of her husband's condition.

Long and persistent care and oversight of each member of the family was arranged through the medical social worker.

The father spent sixty-eight weeks in a sanatorium; but about two years after his discharge he had an acute recurrence, and died. Fred spent one hundred and eight weeks in a sanatorium, and died there within one week of the father's death from tubercular meningitis. The other members of the family are still under medical and social supervision, and are in good condition.

During these six years the mother has received state aid. At the time of the patient's death the care of the two patients in state sanatoria and Mother Aid had cost the public funds $2629. One cannot, of course, estimate what might have been saved in human suffering and in dollars and cents if the little child of four had six months earlier received adequate care, and if, through him, access had been secured to the family.

Case B.—Miss Isabel Gibson, Medical-Social Worker.

N. Z., a Russian, twenty-seven years of age, unskilled laborer, was admitted to the Male Medical Clinic of the Massachusetts General Hospital on March 2, 1920, complaining chiefly of pains in arms and legs, of chilliness, and of insomnia. Diagnosis *myalgia*.

Family history negative, so far as obtained.

Past history essentially negative, so far as obtained. (Language difficulties prevented the securing of accurate or detailed information.)

Physical examination negative, including Wassermann test.

It was obvious that the patient was depressed and apprehensive, and he was referred to the Neurological Clinic on April 6th.

The physician in the Neurological Clinic saw that the social situation was serious, and that it might be the chief factor in the patient's illness. The case was, therefore, referred to a medical-social worker, with diagnosis of *psychoneurosis*, superimposed.

Social Investigation.—As the patient's knowledge of English was very limited, the first step of the social worker was to find a competent Russian interpreter. After considerable difficulty, one was found, and the social worker, accompanied by the

interpreter, visited the patient at his home, and obtained the information from which she was able to supply, for the use of the physicians and for the social work needed, the following history:

Additional Medical History.—For past six months patient has had rather widely distributed pains, localized chiefly in upper and lower extremities. Pains are mostly lancinating. Feeling of chill usually accompanies the pains. Most of his trouble is at night; he feels fairly well in the day. Some frontal headache, especially after a night in which he has not slept well.

Habits.—Excessive cigarette smoking. Alcohol in moderation.

Family History.—Not known, further than that both parents are deceased.

Past Medical History.—Negative, except for pneumonia.

Marital History.—The wife was a frail, thin woman, with a face disfigured by smallpox scars. There were three children, the eldest, a boy of five years; the second, a boy of three and one-half years, and the youngest, a boy of eighteen months. The eldest two—the younger of whom was a mouth-breather, and was said to be subject to "colds"—were pale and delicate, and appeared timid and whimpering. The youngest child seemed more robust than the others, though he also was a mouth-breather.

Occupational History.—Before emigration from Russia patient had been a farmer and had led an active out-door life. For two years after coming to this country he had been employed as a laborer in a foundry, where he had earned good wages, though the work, which consisted in loading pig-iron and carrying coke, was heavy and monotonous. After this, for six months, he was employed in a rubber factory. In the past year he had gone from one temporary job to another, resting for a time after each job. He had recently worked as railroad section-hand, but had found this work very distasteful, and, he thought, too hard for him. He had done no work for two months.

Home.—The family lived in a Russian-Jewish colony, in a fairly good neighborhood. They occupied three rooms on the ground floor; the house was scrupulously clean.

Patient's Character and Mentality.—Education fair, corre-

sponding, probably, to that given in our grammar schools. Speaks fairly correct English. Conscientious; appreciative. Apprehensive; easily discouraged; suggestible; irritable. Self-centered, though devoted to his wife and children.

Social Treatment.—The problem in this case being primarily a social problem, social treatment was, of course, the remedy.

The social worker, having first learned that farm work was what the patient liked, and having satisfied herself that that was the work for which he was best adapted, enlisted the interest of the owner of a large farm about 30 miles from Boston. This man offered the patient employment, and advanced money for the patient's traveling expenses to the farm, and also for the support of the family during his absence.

Meanwhile the social worker brought the wife to the hospital, where a diagnosis of chronic rhinitis was made, and she was relieved by medical treatment. The younger children were also examined, and an operation for removal of adenoids done in each case. The mother was given careful instructions regarding the baby's diet. The worker kept the patient informed of what was being done, and his consent was obtained before the operations were performed on the children.

Three months after the patient's admission to the hospital clinic, he and his family were installed in their new country home, in a cottage which had been built for them on the farm.

Recent reports from the foreman of the farm state that the patient is doing excellent work. The patient, in his letters to the doctor and social worker, writes, "I want to stay here until I die; I am so happy."

It is very significant that the patient has so far overcome the morbid introspection which characterized his mental attitude when he first came to the hospital clinic, that in these letters he never refers to his own health.

While the more difficult work in this case has been completed, the patient and his family will need encouragement and advice until they become well established in their new life.

This case fairly illustrates many cases in which the solution of the social problem is the solution of the medical problem.

Case C.—Miss Elsie Wulkop, Medical-Social Worker.

J. B., an American; age forty-four, minor public official. Married. Admitted to Male Medical Ward October 1, 1919 with question of gastric ulcer; question of neoplasm.

The hospital record states that patient's wife and 2 children are living and well; that the home is a comfortable 8-room house in a suburb of Boston; that good wholesome food is provided, and that there is no economic problem.

Patient's past medical history is given as essentially negative, apart from increasing gastric disturbance for the past year.

Discharged from hospital eleven days after admittance, with diagnosis of *pernicious anemia.*

As the patient was about to be discharged from the hospital the case was referred to the Social Service Department, with a request that the conditions under which the patient worked be investigated, as the patient had said that his office was without direct communication with outside air, and had only artificial light.

The social worker was advised by the physician that the disease was apparently in its early stages, and that its course would probably cover approximately three years. He thought that the patient might, for the present, safely go on with his work. Hospital treatment would, he believed, be necessary from time to time, and the patient might have to be re-admitted within the next three months. The social worker was asked to see that the patient kept strictly to the prescribed diet, and that the level of his general health was kept as high as possible; also that he had plenty of fresh air and rest.

Social Action.—In an interview with the patient the social worker was asked by him to see the head of the division in which patient worked, and ask that the patient's department be transferred to better quarters. On investigation, the social worker found that the working conditions were fairly satisfactory, from the point of view of the patient's health, as the work room was really well supplied with fresh air. She also found that the patient was not under civil service, and was subject to discharge by his superior officer. She decided that it might jeopardize

his position to ask for the change the patient had suggested, and she therefore refrained from taking any action in that respect.

The worker then visited the home (two days after patient's discharge from the hospital) for the purpose of learning what the home conditions were; of conferring with the wife in regard to the patient's physical condition—the seriousness of which the patient himself did not appreciate—and of instructing her in the details of his dietary.

Carefully avoiding any direct questioning on the subject, the social worker elicited all the facts necessary for her purposes regarding the pecuniary circumstances of the family, and the amount which patient would receive as pension in case he became, as he clearly would within a relatively short time, totally incapaciated. The outlook, though normally favorable for a family whose bread-winner was in good health, was not so favorable in view of the medical prognosis.

The social worker, then, must establish such relations of confidence between the family and herself that she would be able to help them plan for the ultimate outcome of the patient's illness, and to assist them in solving the social and economic problems that would arise in the meantime.

A careful medical and social history was obtained by the worker in regard to each member of the family.

The patient was found to be a man of good habits, devoted to his family, prudent, and industrious. He had rather an exaggerated idea of his own importance, which was likely to make him too optimistic, and to lead him into ill-considered action, as was shown in his desire to have the social worker ask for better working quarters, despite the fact that he was very dependent on his position. He was not disposed to be amenable to anyone, except the physician or the social worker, in regard to his own medical care.

The wife was of New England parentage; born and reared on a New England farm. She was an intelligent, well-educated, resourceful, and affectionate woman, whose chief interests were in her family. Apart from extreme defect of hearing she was in good health.

The elder child, a girl of ten years, was subject to frequent "colds in the head," bronchitis, and asthma. She had marked defect of hearing, and was, therefore, handicapped in school work. Though she lacked power of concentration, and seemed somewhat dull, she was thought to be of normal mentality.

The younger child, a boy of nine years, was in good health and of good mentality.

This history the social worker had secured, without obtrusive questioning, at her first visit, at the close of which she was cordially urged to visit the family again.

The medical and social problems, then, were—

To see that the patient reported regularly for medical examination; to see that the prescribed diet was observed; to see that he returned for hospital care when needed.

To secure for the wife expert aural treatment (as her previous treatment appeared to have been of doubtful value).

To secure for the daughter appropriate medical treatment.

The social treatment indicated was to enlist the co-operation of the wife in persuading the patient to plan on a basis of existing conditions, rather than on what he, with characteristic optimism, thought his eventual resources would be; to befriend the family through the patient's illness, and, if possible, before the patient died, to establish the wife and children in a home in Vermont, near her own people, on whom she would be more or less dependent, and who were ready to help her.

Results of Medical-social Treatment Nine Months After Patient's Discharge from the Hospital.—Friendly and sympathetic relations have been established by the social worker with the patient and his family. The patient at first was not at all inclined to adhere strictly to the prescribed diet, and was not responsive to his wife's efforts in that direction. Through the influence of the social worker, however, whose instructions, he knows, come directly from the doctor, he has been held throughout to a careful observance of the hygienic routine prescribed by the physicians. Minor ailments that have developed have

been cared for, and the patient has been kept in the best possible physical condition. He feels so well that he thinks he "will fool the doctors yet."

The daughter is receiving treatment in an aural clinic, with encouraging results. The wife is also under treatment, though the prognosis is not especially promising.

Close relations with the family will be kept up, with persistent efforts toward a satisfactory plan for them when the patient's inevitable breakdown comes.

These cases show that the best interests of the patient demand the correlation of social case work with medical case work, and it is on this principle that social work has been established at the Massachusetts General Hospital. Since its introduction, in 1905, many functions have been assigned to the department—such as medical follow-up work; assisting the administration in determining its policies with public health and other social agencies in the community; teaching pupil nurses the social aspects of their work; interpreting the social aspects of medical practice to medical students; supervising volunteers, and developing occupational therapy. These apportunities have come to the workers in the department in the course of a logical growth. But the fundamental principle from which all phases of hospital social service have developed is that intelligent and skilful social case work is an essential element in successful medical treatment.

The nature of the social problem or, rather, the emphasis on the social elements in the patient's condition, varies in the different clinics—the orthopedic, the children's, the neurologic, the syphilis or tuberculosis clinic—according to whether successful medical treatment depends on a brace or other orthopedic apparatus for which the patient is unable to pay; on the establishment by the patient of new dietary habits; or change of occupation; on making the best of a difficult temperamental handicap, or other unfavorable condition; or perhaps on securing the authority of law for the protection of a child. The underlying principle is always the same; given adverse social conditions, successful treatment depends on a knowledge of the patient's

personality and environment, combined with a knowledge of his physical condition. For the accomplishment of the ultimate purpose of the hospital, therefore, both doctor and social worker are needed.

CONTRIBUTION BY DRS. PAUL D. WHITE AND WILLIAM D. REID

FROM THE CARDIAC CLINIC OF THE MASSACHUSETTS GENERAL HOSPITAL

THE DIAGNOSIS OF MITRAL STENOSIS

Introduction.—Stenosis of the mitral valve is undoubtedly one of the most important of valvular lesions of the heart.[1] This importance is due to the established fact that the same infection which attacks the valvular endocardium to the extent of causing the stenotic change attacks the myocardium as well. The future of such a heart may be said to depend largely upon the amount of impairment the infectious process causes in the cardiac musculature; 25 to 50 per cent. of hearts developing auricular fibrillation (absolute arhythmia) have mitral stenosis.

Granting the importance of mitral stenosis, the desirability for accuracy in its detection and clarity of thought as regards the mechanism and meaning of the condition is obvious. The work in the Cardiac Clinic at the Massachusetts General Hospital during the past year has confirmed the writers in the opinion that there is a considerable variation in the minds of physicians as to what constitutes mitral stenosis. Thus, in cases sent to us previously diagnosed as mitral stenosis, our action has varied from confirmation of the diagnosis, doubt of it, to definite failure to confirm its presence, and in other cases we have been impelled to diagnose the presence of mitral stenosis where not previously detected. Furthermore, the conception of mitral stenosis in much of the literature does not seem entirely satisfactory. Consequently we feel that a discussion of the subject is important.

In seeking for the reasons for the above-mentioned lack of agreement in the diagnosis of mitral stenosis three conditions

* By the term "mitral stenosis" we mean that deformity of the valve which gives rise to a definite stricture.

at once suggest themselves: First, there is a type of heart, the irritable heart, which closely simulates what is described in the literature as early mitral stenosis. Second, there is the thyroid heart, new or old. Third, the murmur of true mitral stenosis shows considerable variation and under some conditions may be absent. Of these conditions we will speak more fully later on.

Explanation of the Murmurs of Mitral Stenosis.—In the normal mitral valve the curtains are well pushed forward, and impede but slightly if at all the blood-stream passing during diastole from left auricle to left ventricle. When stenosis exists the endocarditic changes cause these curtains to adhere to each other, thereby limiting their normal retraction, and in this position they are set into vibration by the stream of blood passing through the valve. The murmur is due to the vibration of the valve curtains and chordæ tendineæ. The intensity of the murmur may be said to depend upon the velocity of the flow of blood and the size of the opening (or, more accurately, the position of the valve curtains). We are not considering for the moment its audibility on the chest surface, in which the thickness of the chest wall, etc., are factors.

The flowing of the blood from the left auricle into the left ventricle during diastole is dependent upon the fact that the pressure is greater in the auricle than in the ventricle. The left intra-auricular pressure is highest late in diastole, when the auricle contracts, and at the beginning of diastole, when the cavity contains the maximum amount of blood; the pressure falls as the auricle is being emptied. This is particularly true of the difference in pressure between auricle and ventricle. The accumulation of blood in the auricles during ventricular systole plus the vacuum produced in the ventricles in early diastole by the elastic dilatation of the ventricular chambers plus gravity are the factors which give rise to the maximal pressure difference between auricles and ventricles in early diastole with the mitral and tricuspid valves open. If auricular contraction is present there is a further high peak (often the highest) in the intra-auricular pressure at the time of auricular systole, *i. e.*, late diastole or pre-

systole. It follows that the velocity of the auriculoventricular blood-stream and therefore the position of the murmurs in diastole is directly dependent upon the variations in left intra-auricular pressure.

As Henderson[1] and others have shown, ventricular filling may be practically complete before auricular systole occurs in a slowly beating heart. Hence even though intra-auricular pressure may be highest at the time of auricular systole, the difference between auricular and ventricular pressures may be less than in early diastole and so slight that the velocity of the blood-stream at this time may be much reduced. This reduction of velocity would account for the absence of a presystolic murmur in mitral stenosis with a slow pulse-rate even with regular rhythm and an earlier diastolic murmur.

As previously stated, the murmurs of mitral stenosis are variable. In relation to time there may be a moderately early or middiastolic murmur with or without a presystolic phase, or finally, the murmur may occupy the entire diastolic period except for a brief interval immediately after the second sound. The diastolic murmur of mitral stenosis does not begin at once with the second sound of the heart, but only after a brief but clearly distinguishable pause. This is due to the fact that the second sound of the heart is produced by the closure of the aortic and pulmonary valves while the mitral and tricuspid valves open a brief interval later. It is only with the opening of the mitral valve and with the definite dilatation of the ventricles that the stream of blood begins its course from left auricle to left ventricle. If the murmur occupies all of diastole there may be an early and a late accentuation, a decrescendo and crescendo. We disagree with the frequent text-book description of the murmurs of mitral stenosis in that we believe that the murmur very rarely occurs *solely* in late diastole or presystole. The pressure in the left auricle may be greatest in early diastole, and in our experience if stenosis of the mitral valve exists to the extent that a murmur occurs with the auricular contraction (presystole) there will also almost invariably be a murmur in early diastole. It is possible that the murmur of early or less advanced mitral stenosis

may even occur in the first half or middle of the diastolic period of the heart rather than in presystole. When auricular fibrillation sets in, the presystolic phase of the mitral diastolic murmur disappears and remains absent so long as the fibrillation persists, due to the fact that the auricles have ceased definite contraction, *i. e.*, they are practically paralyzed. Nevertheless, the early and middiastolic murmur persists so that the diagnosis of mitral stenosis can confidently be made even in the absence of the presystolic murmur.

In quality the murmurs vary from soft to rough. They may often be described as blowing or rumbling. The pitch is usually low and more so than that of any other cardiac murmurs.

Even at its maximum intra-auricular pressure is very low as compared with ventricular systolic pressure. This is a very reasonable explanation of the low pitch or absence of murmurs in mitral stenosis (due to the relatively low velocity of the blood-stream) as compared with mitral regurgitant and aortic murmurs.

The murmur may be absent in mitral stenosis when the velocity of the auriculoventricular stream of blood is low and the stenosis only of slight or moderate degree, as may occur when cardiac failure is present. And in some cases it has been said to be absent when the valve curtains are so rigid from lime deposits, etc., that presumably vibrations sufficient to cause a murmur audible through the chest wall have not been produced (Flint [2]).

Points on Which the Diagnosis of Mitral Stenosis Rests.—The points on which the diagnosis of mitral stenosis rests in the order in which one usually seeks them are as follows:

History.—There is often a history of previous infection of the rheumatic type, *i. e.*, acute rheumatic fever, "growing pains," chorea, tonsillitis, scarlet fever, puerperal sepsis, etc. Its presence is of distinct importance in support of the diagnosis of mitral stenosis, but the latter may be present in a patient with an apparently negative past history. If cardiac disability is present the history may elicit such symptoms as precordial pain, palpitation, dyspnea, and cough on exertion, but these symptoms may occur from heart failure due to any type of heart disease or even to cardiac neurosis with no heart disease at all. Hence

such symptoms are of much less importance in the diagnosis of mitral stenosis than is the past history of rheumatic infection. The history may disclose embolic infarction to the brain or elsewhere and at times hemoptysis. Both of these may be due solely to mitral stenosis and call for careful examination of the heart. The mechanism of the production of this embolism is quite simple. In the large, often sluggish auricles in cases of mitral stenosis, especially where co-ordinate contraction of the auricles has ceased, as in auricular fibrillation, thrombosis is common. Bits of the thrombus or thrombi are apt to be loosened and directed into the blood-stream of the pulmonary circulation in case the embolus comes from a right auricular thrombus, of the systemic circulation (particularly brain and kidneys) if the left auricle is involved. More rarely aphonia from paralysis of the left recurrent laryngeal nerve is caused by mitral stenosis.

Inspection.—There is a facies which is suggestive of mitral stenosis. The face appears a little drawn and there is often a flush, sometimes cyanotic, over the cheek bones, with an underlying sallowness, almost a jaundice. This facies occurs usually only in advanced cases. Cyanosis may be present in the lips and ears, or more generally. The patient is often undernourished in type and the hands and feet are frequently cold. Some clubbing of the fingers is common in patients in whom the disease started in childhood.

The apex impulse, if visible, is usually in the fifth interspace at or near the midclavicular line if the mitral stenosis is uncomplicated. If the heart is considerably hypertrophied it may extend nearly to the anterior axillary line and rarely with extreme preponderant hypertrophy of the right ventricle there may even be a systolic apical retraction in the sixth or seventh interspace well toward or into the axilla. Unusual epigastric pulsation may be found in cases of vertical or "drop" heart (so-called cardioptosis) and of very large forceful hearts due to any cause as well as in chronic mitral stenosis, so that it cannot be taken as evidence of right ventricular hypertrophy.

Palpation over the apex usually locates the impulse in the fifth interspace at or near the left midclavicular line, but further

out or down if the heart is much enlarged. The quality of the impulse varies according to the size, strength, and excitability of the heart, but is rarely heaving or prolonged.

A thrill may accompany the apex impulse. It varies from a mere vibration to a definite purring thrill. It may be systolic, diastolic, or both. In many cases a thrill is absent. In our opinion the importace of the thrill in mitral stenosis has been overstated. It is of no diagnostic value unless well marked and occupying some part of diastole; then it points strongly to the presence of mitral stenosis. But when the thrill is well marked and diastolic in time it will be found that the other evidences of the presence of stenosis of the mitral valve are unmistakable. We have never found the thrill of value in a doubtful case of mitral stenosis.

The pulse is not of much assistance in the diagnosis of mitral stenosis. In a typical case it tends to show a pulse pressure of but slight or moderate degree. In rhythm the mitral pulse is not characteristic. Absolute arhythmia is present if auricular fibrillation has ensued as a complication. The pulse of mitral stenosis is of importance if aortic regurgitation is present, as then the mitral lesion has the effect of distinctly limiting the Corrigan quality of the pulse, *i. e.*, the pulse-pressure is lower than in uncomplicated aortic regurgitation.

Percussion shows the cardiac apex to be in normal position, or if enlargement of the heart is present the border of dulness generally extends laterally rather than downward. Realizing the limitations as to the accuracy of estimating the size of the heart by percussion outlines, we yet believe it to be of value to determine roughly the borders of cardiac dulness. If the convexity of the curve of the cardiac dulness in the left second, third, fourth, and fifth interspaces is outward the presence of a lesion of the mitral valve becomes increasingly probable. This is, of course, merely evidence of prominence of the left auricle which is more accurately determined by Roentgen examination and is the main constituent of the so-called "mitral shape" of the heart. The determination of the curve of the left cardiac border is a simple procedure within the reach of all, and its value

is such that we desire to recommend its use. Increase of the percussion border to the right of the midsternum indicates increase in size of the right auricle. Such a condition is not uncommon in mitral stenosis when auricular fibrillation is present, but inasmuch as such increase can occur with the auricular fibrillation due to other types of heart disease or to pericardial effusions the finding is not of definite value.

E. H. Goodman[3] has emphasized the occurrence of impairment of resonance of the apex of the left lung in mitral stenosis. This has sometimes been associated with hemoptysis, and confusion has arisen as to the presence of pulmonary tuberculosis. It is presumably due to pressure of the dilated left auricle.

Auscultation gives the most decisive evidence on which to base the diagnosis of mitral stenosis. Any of the diastolic murmurs described in the earlier part of this paper may be present. The presence of a diastolic murmur at the apex is positive evidence of organic heart disease, usually of mitral stenosis, but sometimes of aortic regurgitation. At times the murmur is represented by a very low pitched diastolic sound which is better described as a rumble. Sometimes one is in doubt as to whether or not there is a short murmur or an impure third sound. Exercise may help to settle this difficulty. In doubtful cases it may be necessary to adopt means to increase the audibility of the murmurs. Thus the patient should be examined both in the sitting and the dorsal postures. We are in full agreement with H. E. B. Pardee[4] that the murmurs of mitral stenosis are of maximum audibility when the patient leans forward toward the left side, or especially when in the recumbent position and turned on the left side. No examination of the heart is complete unless auscultation is practised with the patient in both the sitting and lying postures. Not infrequently attention to this point will disclose a murmur which would have been passed unnoticed had the patient been examined only when in the sitting position.

Exercise.—Auscultation should be practised after exercise in all suspicious cases. Experience shows that at times the characteristic murmur of mitral stenosis may be audible only

for a few beats after exercise. The inhalation of the fumes of an amyl nitrite pearl has a similar effect in increasing the audibility of mitral stenotic murmurs. Both of the procedures act by increasing the velocity of the blood current passing through the valve.

Still a third point of assistance in detecting the diastolic murmur of mitral stenosis is to use a bell attachment to the stethoscope in auscultation. We have found that apical diastolic murmurs are usually much better appreciated on changing from the diaphragm end-piece to the bell attachment. Auscultation should, therefore, be practised with both end-pieces, since aortic murmurs are better heard with the diaphragm.

The explanation of this last point is of interest and, in view of the emphasis we would like to lay on the value of the bell type of stethoscope in bringing out the murmur, seems worthy of mention here. We cannot do better than to quote from the late Austin Flint[5]: "*All elastic structures, however, have their own vibration times*, and when oscillations are transmitted to them at that rate they are set in motion. This is daily seen in the laboratory, when, as an ill-balanced centrifuge is gradually increased in speed, now one, and now another pipette rack, beaker or burette begins to rattle, and subsides only when the centrifuge's vibrations have altered to become more rapid. It is this which makes it necessary that marching troops break step while crossing bridges, and upon this same fundamental law depends the fact that, if the sound waves of one tuning-fork reach a tuning-fork, string, bell or other sonorous body of similar pitch, the latter is set in action. This law is made of practical use in *analyzing sounds* composed of several tones by exposing a series of resonators to the compound vibration and noting which resonators are set in covibration. It is also of great importance in the construction of stethoscope bells. When dealing with faint murmurs of low pitch, the old Gannet type of stethoscope not infrequently settled an argument, because the large resonating cavity of its bell intensified murmurs of that pitch."

Diastolic murmurs of mitral stenosis are usually heard only over a small area near the apex, generally directly over the point

of maximum impulse or internal to it. In brief, they are most readily detected if one auscults over the apex impulse with the bell form of chest-piece, with the patient lying in the left lateral position after exercise.

The first sound at the apex commonly has a quality described as sharp and snappy, and when of this character should arouse the suspicion of the presence of mitral stenosis. If cardiac failure is present, or if there is a loud systolic murmur of mitral regurgitation masking the first sound, then this sound will not, of course, display the accentuation so often present in mitral stenosis. The presence at the apex of the loud masking systolic murmur in a patient with a history of rheumatic infection should cause one to search for the apical diastolic murmur of mitral stenosis, for it is this type of apical systolic murmur and this type alone that usually spells organic mitral valve lesion.

The second sound at the base may be faint or at the apex absent, but it is usually accentuated over the pulmonic area. Too much weight, however, should not be put on the diagnostic value of accentuation of the pulmonic second sound, since it was shown to exist in 3 per cent. of 17,200 recruits at Camp Grant, Illinois, without mitral stenosis (Pardee[4]).

Reduplication of the second sound is common in the mitral stenotic heart; that of the first sound is less frequent. It is possible that some cases of apparent reduplication of the second sound may be due to the occurrence of an abnormally loud protodiastolic third heart sound. Thayer[6] found a third heart sound in about 65 per cent. of people under forty when they were placed in the recumbent left lateral position. Ordinarily the third heart sound is so faint that it is difficult to appreciate it in nearly so high a percentage. "In mitral stenosis, however, this third heart sound is frequently very distinct. It is apparently due to the sudden tension on the edges of the mitral valve when, with ventricular diastole, it starts to open widely, but cannot. It may well be compared to the opening of a door protected by a chain latch; and the name 'opening snap' is well chosen."[7]

We will discuss the Graham Steell murmur, sometimes present in mitral stenosis, when considering differential diagnosis. It

is appropriate here, however, to speak of the classical publications by Graham Steell on the subject of mitral stenosis. In 1895, years before the clinical condition of auricular fibrillation was understood, Graham Steell wrote a paper which is a model of statistical study. Since it has impressed us as one of the best articles ever written on mitral stenosis, and inasmuch as keen and unbiassed observation permitted the accurate record of murmurs and sounds not then understood (particularly the middiastolic murmur of mitral stenosis), we shall take the liberty of quoting Graham Steell at some length. In the Manchester Medical Chronicle in 1895[17] he described in detail his own auscultatory findings in 60 cases of mitral stenosis collected during a period of twenty-two years; 17 of the 60 cases were proved by post-mortem examination; 3 of the cases probably were not mitral stenosis; Case 43 showed nothing indicating such a diagnosis except on one occasion a presystolic murmur heard not by himself, but by a colleague; Cases 46 and 49 were apparently aortic regurgitation without mitral stenosis.

Steell wrote, " I now subjoin my clinical records, for which I may say I am immediately responsible. It is in no spirit of disparagement of the work of my clinical clerks that I have acted thus, for I have had in view all along the ultimate collation of the results obtained by clinical observation, and such observation, for the purpose indicated, should be the observation of one mind."

Studying the 60 cases Steell found a presystolic murmur without an earlier diastolic murmur at the apex in only 5 (8½ per cent.), while both murmurs occurred together in 27 (45 per cent.), and the diastolic murmur alone at the apex in 16 more (27 per cent). In 9 cases (15 per cent.) postmortem examination showed considerable mitral stenosis, although during life neither presystolic nor diastolic murmurs had been heard. Of these 9, 3 showed loud systolic murmurs at the apex, one of which was inconstant, 3 showed moderate systolic murmurs at the apex, and 3 showed slight systolic murmurs there, two of which were heard only occasionally. Finally, the other 3 cases making up the 60 we have already considered as incorrectly

diagnosed. In this same series the second sound was reduplicated in 39 cases (65 per cent.). The first sound in the mitral area was accentuated in two-thirds of the cases. The Graham Steell murmur was present in only 1 or 2 cases. Auricular fibrillation was probably present in 22 cases (37 per cent.), as determined by measurements on the sphygmograms, all but one of which were published by Steell. He had no knowledge of the condition, however, and, of course, largely missed its association with the disappearance or absence of the presystolic murmur. He found the diastolic murmur alone at the apex in 12 of the fibrillating cases, the presystolic murmur alone in 1 case, and both combined in 6 cases. In these last 7 probably the presystolic murmur which he heard was inconstant and appeared only when the diastolic period was short, or else we have interpreted his radial tracings incorrectly.

One of the reasons for his considering the diastolic murmur heard along the left border of the sternum in cases of mitral stenosis as due to aortic regurgitation and not to pulmonary regurgitation (Graham Steell murmur) was the fact that "by the postmortem records in half the cases the aortic valves showed some change, though often trivial."

With regard to the apical diastolic murmur in mitral stenosis he wrote, "I must confess that I am far from satisfied with my knowledge of this murmur." This was, indeed, a very wise attitude. He also said: "A great deal has been written on the auscultatory signs of mitral stenosis, and there can be no doubt that when the more attractive and characteristic presystolic murmur was first described, the diastolic murmur became, as it were, pushed aside and neglected. Statistics show that it is the more frequent murmur of the two in mitral stenosis." To this we can heartily assent, except in the use of the word "characteristic"; in that we must disagree because of the possibility of confusion between slight mitral stenosis and the forceful or irritable normal heart if we rely too much on a presystolic murmur or thrill.

In a clinical lecture delivered at the Manchester Royal Infirmary in 1898 Steell[17] said that the place of maximum inten-

sity of the characteristic diastolic murmur of mitral stenosis "is not so definitely the apex-beat as is that of the presystolic murmur—it is often quite as well heard to the left of the sternum as high as the fourth cartilage, between the apex-beat and the sternum. Like the presystolic, this murmur is often accompanied by a thrill, best felt at the apex. In rare cases the diastolic murmur of mitral stenosis is widely distributed over the cardiac area, and, as it is usually accompanied by a systolic murmur, the double murmur may exactly resemble the familiar to-and-fro 'double aortic' murmur, or even a friction sound. In one such case, at certain spots in which the second heart sound could be identified, there seemed to be a slight pause between the second sound and the diastolic murmur—at least the murmur did not run off directly from the second sound as the aortic diastolic murmur does when the second sound can be heard along with it [an important observation]. . . . The mitral diastolic is more apt to vary from day to day than the aortic. . . . Another variety of diastolic murmur occasionally met with in cases of mitral stenosis is the murmur I have called the 'murmur of high pressure in the pulmonary artery.' It is well known that in cases of dilated aorta associated with high arterial tension leakage through the strained valves often occurs. Usually such leakage is not permanent at first, but comes and goes. I believe a precisely similar condition occurs in long-standing cases of mitral stenosis as the result of the high pressure maintained in the pulmonary artery, such high pressure being evidenced by the greatly accentuated pulmonary second sound. . . .

"There is no pulse pathognomonic of mitral stenosis. In the presence of symptoms, the most common pulse observed is that described as of the second stage." Steell described under the second stage the condition of auricular fibrillation (absolute irregularity), at that date not recognized as such. He failed to make one observation made later by Mackenzie—*i. e.*, the disappearance of the presystolic phase of the diastolic murmur in auricular fibrillation, for he says, "There does not seem to be any definite association between the pulse and any auscultatory sound, though naturally the first stage pulse (regular) is most

often associated with a presystolic murmur." Here he did suggest that there may be some slight association.

x-Ray.—Roentgen evidence consists mainly in the demonstration of the "mitral shaped heart," in which the organ appears more rounded than normally. The enlargement is lateral, and particularly in the region of the auricles.

Electrocardiogram.—The "typical" tracing may show the auricular or *P* wave over 3 mm. in height or over 0.1 sec. in duration, which is evidence of auricular hypertrophy, most commonly found in mitral stenosis; also it may yield evidence of right ventricular preponderance. The absence of both these changes does not disprove the presence of mitral stenosis, inasmuch as it is only the chronic advanced cases of uncomplicated mitral stenosis that give the "typical" electrocardiogram. In other words, if the electrocardiogram shows these findings we may be fairly sure of the presence of mitral stenosis; if it does not, it is of no help. Of course, if the auricles are fibrillating the *P* wave will be absent.

Differential Diagnosis.—There are just three conditions in which there is much reason for confusion as to the presence of mitral stenosis. These are a certain type of the normal heart, the thyroid heart, and that in which aortic regurgitation is accompanied by the Austin Flint murmur at the apex. We shall discuss these in more detail.

With the aid of Miss Ruth E. Lewis we have made a brief statistical study of the "mitral stenosis" cases sent to the Cardiac Clinic of the Out-Patient Department of the Massachusetts General Hospital from the other clinics during the past winter and spring; 36 cases were referred with the diagnosis of "mitral stenosis." After special study we confirmed the diagnosis in 26, changed it to "cardiac neurosis" in 4, to "rheumatic heart disease with mitral involvement" in 2, to "aortic regurgitation" in 1, to "thyroid heart" in 1, and to "no heart disease" in 2, one of whom showed ventricular premature beats.

The normal heart sometimes presents a picture which closely simulates that which is, in our opinion, often erroneously described as early mitral stenosis. Under this heading we may

include the heart temporarily irritable or excited for some reason, and the heart of the cardiac neurotic which was so wide-spread during the war—the "soldier's heart." Symptoms and signs of excessive nervousness should help one to differentiate this condition from doubtful mitral stenosis. Sometimes, however, the two conditions are combined.

It has been shown that the first sound of the heart—*i. e.*, the systolic sound—is preceded normally by a short presystolic sound due to auricular systole (Bridgman[8]). Inasmuch as these sounds are continuous and practically synchronous with systole, so far as the usual clinical methods of examination go, an increase in the force of the presystolic phase cannot be distinguished from the beginning of a rather forceful first sound. Attempts to interpret such an increase differently are sure to lead one astray. It is wiser, therefore, as White[9] has already insisted upon, to regard as presystolic murmur only that which is clearly diastolic in time or associated with an early or middiastolic murmur. The same policy holds true with regard to the presystolic thrill. Henry Sewall[10] writes: "In the structurally normal heart, especially in conditions of excitement, the first sound frequently begins with a crescendo tone, simulating closely the faint and brief presystolic murmur or acute accent initating the first sound in certain stages of organic mitral stenosis." Thacher[11] finds this presystolic murmur occurring in the normal heart especially common in cases where the chest is depressed near the nipple by an old Harrison groove. Morris and Friedlander[12] state that "a presystolic thrill, best felt in the erect posture, when the heart's action is accelerated through emotion or exercise, associated with reduplication of the first sound and more or less systolic shock at the apex, becoming less perceptible or disappearing when the patient is in the recumbent position . . . is a normal phenomenon in a certain proportion of healthy adults."

In cases with a thin chest wall and especially when the velocity of the blood-stream flowing through the mitral valve is accelerated, as by excitement or exercise, the thrill, murmur, and peculiar quality of the first part of the first sound at the apex may become apparent by auscultation on the chest wall.

It thus becomes evident that it is dangerous to place much value on the occurrence of a presystolic thrill or murmur at the apex. As already stated, we consider that in true mitral stenosis a presystolic murmur rarely ever occurs without a murmur or rumble in the earlier part of diastole. Therefore we would again emphasize that in the diagnosis of stenosis of the mitral valve attention should be centered on the detection of the findings in diastole, and a halt may well be called upon the often difficult feat of timing a thrill or murmur as presystolic rather than as synchronous with or masking the first sound.

The thyroid heart, through its increased force, may resemble closely the irritable or soldier's heart, and so may be readily confused with mitral stenosis if care is not used. The auricular fibrillation occurring in advanced cases may sometimes be difficult to distinguish from the fibrillation of the mitral stenotic heart. The presence of a diastolic rumble at the apex will tell us that mitral stenosis is present and that the heart disease is rheumatic and not thyroid. A loud blowing systolic murmur masking the first sound at the apex and a past history of rheumatic infection also help very much in differentiating doubtful mitral stenosis from the irritable and the thyroid heart. Of course hyperthyroidism usually is attended by hypertrophied thyroid gland and exophthalmos.

Aortic regurgitation accompanied by the murmur of Austin Flint[13] at the apex gives rise to a most difficult problem of differential diagnosis from organic mitral stenosis. The Flint murmur is sometimes called the murmur of functional mitral stenosis. It is produced by the stream of blood entering the left ventricle from the left auricle through the mitral valve narrowed by the pressure on its anterior cusp of the regurgitant stream descending from the aortic valve. Thayer[14] reported its occurrence in 56 per cent. of cases of pure aortic regurgitation. The same authority finds the Flint murmur occurring in any stage of diastole and at times accompanied by a thrill. Therefore it cannot be differentiated *per se* from the murmur of true mitral stenosis. One must look to the accessory signs for assistance in this differential diagnosis. Thus, in mitral stenosis as contrasted

with regurgitation through the aortic valve, the apex impulse appears to hit the chest wall a sharp tap, and withdraws quickly rather than giving the sensation of a powerful heave which takes more time to withdraw; the first sound tends to be sharp and snappy rather than loud and booming, and the pulse is small rather than Corrigan in type. Blood-pressure determination shows a normal or rather low pulse-pressure* in uncomplicated mitral stenosis and a high pulse-pressure in uncomplicated aortic regurgitation which is of sufficient degree to give an Austin Flint murmur. When organic mitral stenosis complicates aortic regurgitation there is a very definite tendency for a lessening of the high pulse-pressure of the latter lesion. Consequently it is always important to determine the pulse-pressure in these doubtful cases. The finding of the mitral shape in the Roentgen examination and of evidence of preponderance of the right ventricle in the electrocardiogram point toward the presence of organic stenosis of the mitral valve. Aortic regurgitation uncomplicated is often associated with left ventricular preponderance by electrocardiogram and a longer heart by *x*-ray. The ventricular hypertrophy as shown by *x*-ray and electrocardiogram is generally balanced where both lesions are present together.

The diastolic murmur of aortic regurgitation should cause little confusion with that of mitral stenosis because of its location along the left border of the sternum, its more blowing quality, its higher pitch and its earlier occurrence, coming with, sometimes masking, the second sound, or immediately following it. In mitral stenosis it will be remembered that the murmur commences at a definite interval after the second sound.

The Graham Steell murmur in cases of mitral stenosis is, in our experience, relatively uncommon, though Cabot[15] has stated that it is often present in mitral stenosis with sound aortic and pulmonary valves, and Goodman[16] reports its presence in 33 per cent. of a series of cases. Graham Steell himself reported it as a rare murmur.[17] It is an early high-pitched diastolic murmur

* The pulse-pressure is the difference between systolic and diastolic pressures.

best heard at the third left costal cartilage, and is generally thought to be due to functional regurgitation through the pulmonary valve due to increased pressure in the pulmonary artery. It is to be distinguished from the murmur of aortic regurgitation, although its time and position and quality are apparently identical. The distinction rests then on the blood-pressure and pulse findings, which in the case of aortic regurgitation possess the Corrigan characteristic, on the *x*-ray and electrocardiographic findings, which are those of right ventricular hypertrophy if the murmur is a Graham Steell. On account of the differences of opinion regarding this murmur we must maintain an open mind concerning it until we have more convincing proof of its frequency. It seems likely that it is sometimes said to occur when actual aortic regurgitation is present associated with mitral stenosis.

Both aortic and mitral valves may be attacked in rheumatic heart disease, whereas if syphilis is the cause of the cardiac lesion the aortic is the valve involved. However, we believe that in spite of all care in examination of the heart it is not unusual to meet cases in which a wholesome doubt remains as to whether organic mitral stenosis is complicating an aortic regurgitation or the findings are due to the functional stenosis of Flint.

Classification.—The terminology with regard to mitral valve lesions is not very satisfactory. One hears of mitral disease, of double mitral disease, of mitral regurgitation, of mitral stenosis. Some claim that every case of organic mitral regurgitation has also some stenosis. The best method of classification is to describe the condition as "rheumatic heart disease with mitral involvement" if there is no apical diastolic murmur and if the apical systolic murmur is very loud and masks the first sound. There may or may not be slight stenosis—there is no proof of it on physical examination. If an apical diastolic murmur is heard we can speak of "rheumatic heart disease with mitral stenosis," whether or not there is definite past history of rheumatic infection. Evidences of much or little stenosis may be present, but it is notoriously difficult to judge the degree of stenosis by the intensity or quality of the murmur. With the

onset of auricular fibrillation, which eventually comes to many cases of mitral stenosis, we have a change in signs, but it s still possible to make the second diagnosis above, adding to it "and auricular fibrillation." Then, finally, we have signs of "heart failure of the congestive type" to add to the previous diagnosis whether auricular fibrillation is present or not. Auricular flutter and paroxysmal tachycardia may occur in mitral stenosis also. Rheumatic infection is almost the sole cause of chronic change in the mitral valve; hence, always the initial words of the diagnosis should be "rheumatic heart disease."

Case reports illustrating the most important points discussed above will follow.

Summary.—Mitral stenosis is an important complication of rheumatic heart disease which necessitates differentiation from the irritable heart, the thyroid heart, and the complex due to aortic regurgitation.

The important points in the diagnosis of mitral stenosis are the rheumatic history, the diastolic rumble at the apex, the "mitral shape" (prominent auricles) of the heart outline by percussion and *x*-ray, at times the "mitral facies," the history of cerebral or other embolism in a relatively young individual, and the electrocardiographic findings of auricular hypertrophy and right ventricular preponderance. The presystolic murmur and thrill are less pathognomonic of mitral stenosis than the middiastolic murmur, and if doubtful or if not supported by the latter it is usually unsafe to place reliance upon them. Mitral stenosis can be diagnosed in the absence of the presystolic murmur.

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CASES

GROUP A.—DEFINITE MITRAL STENOSIS

Case. 1.—M. G. H. O. P. D.* No. 372,170. March 8, 1920. Single man, aged twenty-four, employed in an iron foundry, born in Russia.

Sent to Cardiac Clinic for examination as to cause of pain in chest. This pain was across upper sternal region and there was slight palpitation on exertion.

Past History.—No definite history of a rheumatic infection.

Present History.—Dyspnea, dizziness, precordial pain, and palpitation for six months.

Physical Examination.—Heart slightly enlarged transversely by percussion. Diastolic and presystolic murmurs with thrill at apex. Slight diastolic murmur audible along left sternal border. This latter murmur does not occur in early diastole. Blood-pressure: systolic 110, diastolic 80. Tonsils large, probably an etiologic factor.

Electrocardiogram: Sino-auricular tachycardia, high *P* wave and right ventricular preponderance (in accord with mitral stenosis). (See Fig. 45.)

Diagnosis.—Rheumatic heart disease with mitral stenosis.

Discussion.—The explanation of the slight diastolic murmur audible along the left sternal margin is of interest. The low

* All the cases reported are Out-Patient Department cases at the Massachusetts General Hospital. Nearly all the abstracts summarize the findings and diagnosis in other departments before the cases were referred to the Cardiac Clinic.

pulse-pressure and electrocardiographic findings do not favor regurgitation through the aortic valve as the cause. It is not the Graham Steell murmur, since the latter commences with the second sound. It is probably the same murmur as that at the apex. Occasionally one hears it nearer or closer to the sternum as well as at the apex.

The electrocardiogram is quite typical of mitral stenosis.

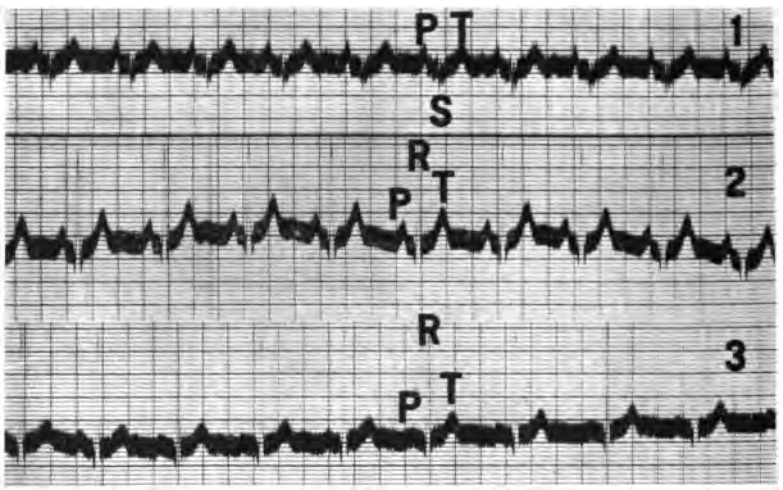

Fig. 45.—Electrocardiogram of Case 1, No. 372,170, showing high auricular or *P* wave in Leads 1 and 2 and right ventriclar preponderance. Normal rhythm. Mitral stenosis. Abscissa = 0.2 sec. Ordinate = 10^{-4} volt. Leads 1, 2, and 3 so labeled. *P* = auricular complex, *R-S* and *T* = ventricular complexes.

Case 2.—M. G. H. O. P. D. No. 366,036. April 26, 1920. Married woman, aged thirty-two, housewife, born in Russia.

Family History.—Husband and 3 children living and well. No miscarriages. One sister has heart trouble.

Past History.—Feet, knees, and hands were swollen at sixteen years. In bed for eight weeks. Typhoid fever at seventeen years.

Present Illness.—Dyspnea and weakness for past few months. Does her own housework, but believes it too arduous. Can

walk one-half mile or more, but becomes short of breath if she goes too fast. One flight of stairs causes dyspnea. Tires easily.

Physical Examination.—Diastolic murmur with presystolic roll ending in sharp slapping first sound at apex. Heart action tumultuous, rate 89, regular.

May 3, 1920: Examination now shows the diastolic murmur to occupy entire diastole of the heart, except for a very brief interval in early diastole.

Diagnosis.—Mitral stenosis and regurgitation. Rheumatic heart disease.

Discussion.—A typical case of mitral stenosis with regular rhythm and showing some variation in the diastolic murmur. The presystolic roll, it will be noted, is preceded by a murmur occuring earlier in diastole.

Case 3.—M. G. H. O. P. D. No. 307,342. August 14, 1916. Male, forty years old, married, blacksmith, born in Russia.

Chief complaint is pain across epigastrium, mostly when working. Duration one year. This pain has no relation to meals.

Physical Examination.—Heart not enlarged to percussion. Sounds rather distant, with "moderate irregularity" and some reduplication of the second sound at base.

Diagnosis.—Constipation.

Feb. 9, 1918: Irregularity of heart has continued; no murmurs heard.

Apex rate,	120
Radial rate,	70
Deficit,	50

Digitalis prescribed.

Dec. 22, 1919: Referred to Cardiac Clinic.

Past History.—Recalls no rheumatic infection or serious disease of childhood.

Present Illness.—Dyspnea for many, many years, ever since he can remember anything. Worse for the past four years. No pain now.

Physical Examination.—Absolute arhythmia. Middiastolic

blow with slight thrill at apex. With rapid heart-beats the murmur ends at the first sound. No presystolic murmur. Question of a systolic murmur at apex. No murmurs at base. Heart enlarged to percussion.

Apex rate, 90
Radial rate, 64
Deficit, 26

Diagnosis.—Rheumatic heart disease, mitral stenosis, auricular fibrillation.

Dec. 21, 1919: *x*-Ray report. Heart shadow shows general enlargement, is somewhat triangular in shape, and the curves of the various chambers are indistinct. Shadow of great vessels indistinct in both diameters. Findings could be due to an enlarged and dilated heart or to adhesive pericarditis. Heart border is 5.8 cm. to right of median line and 10.6 cm. to left. Total transverse diameter is 16.4 cm. Length of heart is 18.2 cm. Transverse diameter of great vessels is 6.2 cm.

Dec. 29, 1919: Middiastolic murmur is almost impossible to hear today.

Discussion.—The above is a definite case of mitral stenosis with auricular fibrillation as a complication. It is probable that the cardiac irregularity noted on the patient's first visit was due to auricular fibrillation. The variability of the murmur should be noted. The Roentgen report introduces a question as to the presence of adhesive pericarditis, but in no way should shake the diagnosis of mitral stenosis. This patient is getting along very satisfactorily under digitalis therapy.

Case 4.—M. G. H. O. P. D. No. 406,978. Feb., 1920. Male, thirty years old, married, printer, born in Massachusetts.

Chief Complaint.—Shortness of breath, nausea, and pounding in the epigastrium for the past six months.

Past History.—Appendectomy twenty years ago. Rheumatic fever ten years ago, lasting three weeks.

Present Illness.—Slight dyspnea first noticed three or four years ago. Was recently a patient in the hospital wards for treatment of cardiac failure with edema. Summary of hospital

record: "Discharged April 25, 1920. Chronic endocarditis. Decompensation. Auricular fibrillation. Pleurisy with effusion. Digitalized with tincture of squill and later with digitalis, with very rapid and striking improvement. Renal function 45 per cent. Electrocardiogram showed auricular fibrillation and probable right ventricular preponderance (Fig. 46). Wassermann reaction negative."

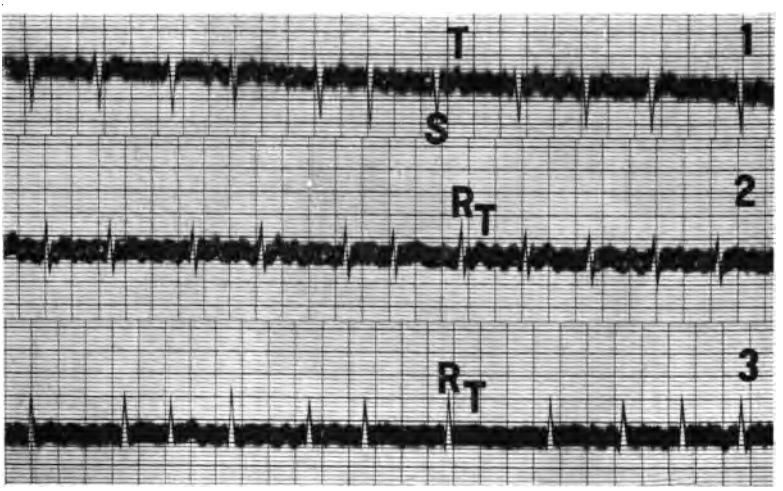

Fig. 46.—Electrocardiogram of Case 4, No. 406,978, showing auricular fibrillation and right ventricular preponderance. Mitral stenosis (more advanced than in Fig. 45). Abscissa = 0.2 sec. Ordinate = 10^{-4} volt. Leads 1, 2, and 3 so labeled. P = auricular complex, R-S and T = ventricular complexes.

May 10, 1920: Reported to Cardiac Clinic. Pulse 72, absolutely irregular. Slight cyanosis. Mitral facies. Systolic and middiastolic murmurs at apex. Apex impulse in fifth space to left of midclavicular line.

Apex rate, 70
Radial rate, 62
Deficit, 8

Has been taking two tablets of digipuratum daily and is feeling fine.

Diagnosis.—Rheumatic heart disease, mitral stenosis, auricular fibrillation.

Discussion.—A typical case of rheumatic heart disease with mitral stenosis and auricular fibrillation. No presystolic murmur.

Group B.—Doubtful Mitral Stenosis

Case 5.—M. G. H. O. P. D. No. 362,287. June 8, 1918. Boy, aged twelve years, in school, born in Massachusetts.

Sent to hospital by a local physician as case of chorea and endocarditis. Was in the ward from June 18 to July 23, 1918. Diagnosis: Chorea; endocarditis (mitral stenosis).

Aug. 26, 1918: Heart: percussion outlines 2.5 cm. to right and 9 cm. to left of midsternum. Loud systolic murmur all over precordia. Pulmonic second sound equals aortic second, which is accentuated.

Jan. 9, 1919: Improved. Twitches only occasionally. No cardiac symptoms. Physical examination shows heart as before.

April 12, 1920: Referred to Cardiac Clinic. Pulse 72, regular; weight 92 pounds.

Present Illness.—Getting along well. Twitching (old chorea) is almost gone. Goes to school and plays like other boys.

Physical Examination.—Apex impulse lifts the sixth rib in nipple line. Blowing systolic murmur at the apex transmitted to axilla and masking the first sound. Pulmonic second sound accentuated. Question of middiastolic murmur at apex after exercise. Slight choreic movements of shoulders.

x-Ray: Heart and great vessels appear to be within normal limits. Auricles slightly prominent. The shape of the heart consistent with early mitral stenosis.

Diagnosis.—Chorea; rheumatic heart disease, with mitral involvement.

Discussion.—In the presence of so positive a history of chorea it does not seem safe to ignore the loud systolic murmur at the apex. The Roentgen report is "normal," but this statement is modified by "auricles slightly prominent," which again suggests mitral disease. The failure to elicit a definite diastolic murmur

or rumble makes a positive diagnosis of mitral stenosis impossible. This boy is living a normal life and has been advised to report to the Cardiac Clinic at intervals for examination.

Case 6.—M. G. H. O. P. D. No. 298,998. May 19, 1916. Girl, seventeen years old, in school, born in Massachusetts.

Discharged from hospital ward March 31, 1916. "Mitral stenosis. Premature ventricular systoles. Wassermann reaction negative. Electrocardiogram shows prolongation of the *P-R* interval, slight grade of heart-block. Given strict directions about limiting activity."

Family History.—Mother had "heart trouble."

Past History.—Children's diseases. Chorea since age of eleven, last attack one year ago. Recent severe tonsillitis.

Present Illness.—Duration five months. Tonsillitis with "valvular heart involvement." Has been in bed for two months and confined to the house since then. Palpitation is the chief symptom. Comes to clinic to report as to condition.

Physical Examination.—Thin. Small cryptic tonsils. No glandular enlargement. Heart rate 96, measures by percussion 3 cm. to right and 12.5 cm. to left of midsternum. Occasional "dropped beat." First sound sharp and valvular. Harsh systolic murmur with a "faint presystolic rumble."

Urine: Normal color, cloudy, acid reaction, specific gravity 1019, albumin trace, sugar absent. Sediment; rare hyaline cast, rare red blood-corpuscle.

June 20, 1916: Tonsillectomy. Urine shows the slightest possible trace of albumin.

April 26, 1920: Referred to Cardiac Clinic.

Present Illness.—Feels well, no symptoms. Has worked as a stenographer for the past three years. Comes to clinic in response to a letter of inquiry as to her present condition.

Physical Examination.—Heart: Apex impulse in sixth space, midclavicular line. Blowing diastolic murmur along left border of sternum. At apex systolic murmur and low-pitched middiastolic rumble with presystolic accentuation. Blood-pressure: systolic 124, diastolic 60.

Opinion.—Rheumatic heart disease, aortic regurgitation, mitral stenosis (question of functional, *i. e.*, Austin Flint).

Discussion.—The location of the apex impulse in the sixth interspace added to the well-defined diastolic murmur audible along the left sternal border make the diagnosis of aortic regurgitation certain. To account for the diastolic blow at the sternal border as that of Graham Steell does not explain the cardiac enlargement downward. The pulse-pressure does not help us much, save to increase the probability of the presence of a true mitral stenosis, which lessens the higher pulse-pressure of an uncomplicated aortic regurgitation. Roentgen and further electrocardiographic examinations may give more assistance in settling the doubt.

Case 7.—M. G. H. O. P. D. No. 323,386. Feb. 27, 1917. Female, twenty-nine years old, married, housewife, born in Italy.

Complaint.—Swelling all over body of three months' duration.

Family History.—Father died at forty-five years of an accident, mother has heart trouble and diabetes. No history of tuberculosis, cancer, or mental diseases in the family. No history of venereal disease in husband.

Past History.—Measles, diphtheria, tonsillitis often, one miscarriage at three months. Denies venereal infection. Catamenia regular and painless.

Present Illness.—About Christmas, 1916 fainted while talking to her sister. Since then has had marked dyspnea on slight exertion. Very weak. Two weeks ago edema became so marked that patient had to go to bed. Palpitation on slight exertion. Cough accompanies the dyspnea and palpitation.

Physical Examination.—Heart borders by percussion 5 and 13 cm. "Presystolic murmur transmitted to axilla." Systolic murmur at apex transmitted over precordia. Pulmonic second sound plus and greater than aortic second. Legs show some edema, as indicated by marks from creases in stockings. No cyanosis on lying down. Tonsils irregular, moderately enlarged.

Diagnosis.—Mitral stenosis and regurgitation.

Patient has made many visits since then. No essential change noted. Never definite edema.

April 26, 1920: Reported to Cardiac Clinic.

Present Illness.—Very nervous during the past few years. Faints occasionally. Frequently dyspnea on exertion and with one pillow. Palpitation on exertion. Occasionally slight swelling of hands and feet.

Physical Examination.—Apex impulse fifth space, outside midclavicular line. Blowing systolic murmur at apex, continuous with the first sound. Question of slight middiastolic murmur or sound. Pulmonic second sound much accentuated.

℞. Roentgen mensuration of heart.
Electrocardiogram.

Opinion.—Probably rheumatic heart disease with mitral involvement, plus cardiac neurosis.

May 3, 1920: Electrocardiogram: Normal rhythm, no abnormalities. Roentgen report: Heart shadow is apparently slightly increased in transverse diameter and it is rather round in shape. Diagnosis deferred. Measurements: Right 4.5 cm., left 8.2 cm., total transverse 12.7 cm., length 14.3 cm., breadth 10.6 cm., internal diameter of chest 24.4 cm.

Feels well, sole complaint is that she is weak and nervous at times.

Physical examination as of April 26th. No diastolic murmur appreciated.

℞. Diagnosis of cardiac neurosis emphasized. To return in six months for re-examination. Referred to Social Service Department.

June 7, 1920: Report from social worker that patient attempted suicide by drinking poison. Reason for the above action said to have been domestic trouble and to have no relation to cardiac condition.

Discussion.—The symptoms are mostly those associated with cardiac neurosis; the attempted suicide shows the mentally unstable type of individual. The location of the apex impulse outside the midclavicular line, the slight changes reported in the Roentgen examination, the markedly accentuated pulmonic

second sound, and the history of tonsillitis makes us strongly suspect that the systolic murmur at the apex is associated with rheumatic heart disease with mitral involvement. In the absence of a more definite diastolic murmur we cannot positively confirm the previously made diagnosis of mitral stenosis.

Case 8.—M. G. H. O. P. D. No. 402,533. Dec. 15, 1919. Married woman, thirty-three years of age, housewife, born in Ireland.

Present Illness.—Chief complaint is cough of two weeks' duration, accompanied by yellow sputum. Appetite good, bowels move with laxative. No pain, no fever.

Physical Examination.—Weight 149 pounds, temperature 98.6° F., pulse 120. Heart: Presystolic murmur in sixth space beyond midclavicular line. Pulmonic second sound accentuated.

Diagnosis.—Mitral stenosis.

June 7, 1920: Referred to Cardiac Clinic.

Past History.—Never rheumatic type of infection. Always worried and nervous.

Present Illness.—Palpitation for four or five years, worse for past six months, especially after excitement. No dyspnea except on climbing steep stairs. No precordial pains.

Physical Examination.—Pulse 77, regular; weight 145.5 pounds. Heart: Loud first sound, systolic murmur at apex, reduplicated second sound, all increased by exercise. No diastolic murmur heard.

Electrocardiogram: *P* wave high (+3), question of slight right ventricular preponderance. Normal rhythm.

Discussion.—In our opinion this case is probably cardiac neurosis. The findings in the electrocardiogram favor mitral damage, however, so that for the present we must regard the condition with some doubt. An *x*-ray examination may give further help, although in early mitral stenosis little abnormality occurs in the heart shadow.

GROUP C.—CASES INCORRECTLY DIAGNOSED MITRAL STENOSIS

Case 9.—M. G. H. O. P. D. No. 379,283. Feb. 20, 1919. Married woman, thirty-eight years old, housewife, born in Ireland.

Family History.—Negative.

Past History.—Complete hysterectomy eleven years ago, and since then health has been poor. During the past three to four years she has had some dyspnea on ascending stairs. "Stomach is weak" and occipital headache is frequent. Bowels require cascara to combat constipation. Recalls no diseases.

Chief complaint is soreness of neck radiating to left shoulder. Five days ago neck began to swell and motion of neck and left arm became painful.

Physical Examination.—Pupils react well to light and distance. Eyes bulge slightly, with staring expression. No glands in neck. Heart borders by percussion 3 and 10.5 cm. to right and to left of midsternum respectively. Apex impulse palpable in fifth space, accompanied by a thrill. "Presystolic murmur" at apex and soft blowing systolic murmur at aortic area. Aortic second sound much accentuated. Pulse 80. Weight 114 pounds. Blood-pressure: 270 systolic, diastolic 190.

Wassermann reaction negative.

Diagnosis.—Hypertension. Arteriosclerosis. Mitral stenosis.

March 20, 1919: Dyspnea variable. Feels quite nervous, palms moist, thyroid palpable. Urine: No albumin, no sugar.

℞. Metabolism test.

x-Ray report, May 21, 1919: "Specific aortitis. Heart border is 5.5 cm. to right of median line and 8. 5 cm. to left. Total transverse diameter is 14 cm. Length of heart is 15.5 cm. Base is 11 cm. Total transverse diameter of great vessels is 9.5 cm."

May 29, 1919: Referred to South Medical Department for treatment of luetic aortitis. No history of primary or secondaries. Never pregnant. Husband living and well.

Patient was treated with mercury and iodids without apparent benefit. On Aug. 12, 1919 she was returned to the Female Medical Department.

Nov. 3, 1919: Referred to Cardiac Clinic.

Heart shows no murmurs. Apex impulse felt in sixth space outside midclavicular line. Aortic second sound much accentuated. Blood-pressure: systolic 216, diastolic 130. Pulse 80; weight 115 pounds.

Opinion.—No evidence of mitral stenosis or primary heart disease.

Nov. 10 to 19, 1919 patient was in ward. Blood-pressure 210/140 and 170/140. Phthalein test 60 per cent.; no fixation of the specific gravity of the urine in two-hour test; blood chemistry normal. Wassermann test negative.

Diagnosis.—Precocious atherosclerosis, hypertension, nephritis, sclerotic arch.

Jan. 20, 1920: Cardiac Clinic. Vital capacity, 2400 c.c. Four months ago is said to have had a visible swelling of the thyroid gland persisting about one week.

℞. Basal metabolism estimation.

Jan. 21, 1920: Basal metabolism (determined in laboratory of Dr. J. H. Means) equals plus 36 per cent. above normal. Dr. Means writes: "She has symptoms which certainly are very suggestive of early Graves' disease, and the increased metabolism makes this seem a reasonable diagnosis. I should not start her on the *x*-ray treatment at once, but would rather keep on with rest and add quinin hydrobromid."

Pulse 80; weight 116 pounds.

Feb. 9, 1920: Is sure she is better. With the shortness of breath there is a smothering feeling located under the upper end of the sternum.

Physical Examination.—Pulse 73; weight 116 pounds. Soft systolic murmur at base of heart and transmitted outward to the right. Blood-pressure: systolic 210, diastolic 140.

x-Ray mensuration of heart: Right 4.5 cm., left 9.3 cm., transverse 14 cm., length 16.3 cm., breadth 10.4 cm., great vessels 8.2 cm.

May 24, 1920: Basal metabolism is now +19 per cent. Patient has used the triple bromids during the past six weeks.

Discussion.—The abnormality of the thyroid apparently

attracted some attention at the time of the first visit, and on March 20, 1919 the metabolism test was requested. That the latter was not obtained was presumably due to the war conditions. In view of the marked broadening of the great vessels, as shown in the *x*-ray report of May, 1919, the patient was given tentative antisyphilitic therapy. The second *x*-ray report, February, 1920, shows a diminution of 1.2 cm. in the width of the aortic shadow. The drop in blood-pressure from its very high figure on the first examination may have something to do with this change, but also it is quite likely that the first plate showed some distortion and so gave too great a figure.

From the data on the record the diagnosis of nephritis does not seem tenable. Hyperthyroidism is proved and hypertension frequently accompanies hyperthyroidism. Evidence of primary valvular disease of the heart is wanting.

Case 10.—M. G. H. O. P. D. No. 305,471. July 24, 1916. Male, single, twenty-seven years old, carpenter, born in Italy.

Family History.—Negative.

Past History.—"Rheumatism" fourteen years ago.

Present Illness.—Complains of rheumatism, duration four months. Pain in all the joints. Sweats at night and has a cough.

Physical Examination.—Heart not enlarged, action regular. "Presystolic murmur" heard at the apex. Otherwise negative.

Diagnosis.—Subacute arthritis, mitral stenosis.

May 27, 1917: Dyspnea and palpitation when working. Edema of ankles at times. No rheumatism since last visit. Heart: Slight enlargement to 2 cm. to left of nipple line. First sound of poor quality. Soft-blowing systolic murmur immediately following first sound and "replacing the second sound" (sic). Slight systolic murmur over the aortic area, not transmitted. No thrill, no edema.

April 26, 1920: Referred to Cardiac Clinic.

Present Illness.—Three months ago acute rheumatic fever. Before that he had been in very good condition for years. Eleven hours of work daily. Still has some pain in joints and over precordia.

Physical Examination.—Pulse of slight waterhammer type. Blood-pressure: systolic 125, diastolic 60. Apex impulse felt in fifth interspace inside nipple line. Early diastolic murmur heard along left border of sternum and at apex. No presystolic accentuation. Soft systolic murmur at apex.

Opinion.—Rheumatic heart disease, aortic regurgitation. No evidence of mitral stenosis.

℞. Wassermann reaction.
x-Ray, 7-foot plate.

Discussion.—The occurrence of a presystolic murmur, as on the first examination, is unsafe evidence on which to base the diagnosis of mitral stenosis. Later at the time we examined this patient there was definite evidence of aortic regurgitation and, in view of the positive history of rheumatic infection, the latter is the probable etiologic factor. The patient has not yet returned for the Wassermann test or *x*-ray.

Case 11.—M. G. H. O. P. D. No. 323,825. March 23, 1917. Single man, twenty-four years of age, shipper in drug concern, born in Massachusetts.

Past History.—Pneumonia at eight or nine years, scarlet fever, measles; denies venereal disease.

Complaint.—Pain in left side of chest. Feels tired. Last April weighed 149 pounds. Dry cough, which patient attributes to dust from his work.

Physical Examination.—Many decayed teeth. Heart: "Presystolic murmur and thrill" in sixth space, nipple line. Pulmonic second sound accentuated. Slight increase in cardiac area to left. Pulse rises from 80 to 96 on exercise. Weight 139 pounds.

Urine normal.

Diagnosis.—Mitral stenosis.

May 17, 1920: Referred to Cardiac Clinic. Comes in response to letter of inquiry as to his condition.

Past history as above save for pneumonia in December, 1918.

Present History:—No complaints. Dates heart trouble to March, 1917, when, he states, he came to hospital for general examination. Three flights of stairs and walking never cause

shortness of breath. Occasionally has slight discomfort in left lower axilla. Aug. 5, 1918 to July 18, 1919 served in the motor transport service of the U. S. Army. No trouble with his heart. Noted that his heart was subjected to particularly careful examination both on his admission and on his discharge from the army. Works daily in "parts department" of an automobile concern. Cigarettes 8 to 10 per day and 3 cups of coffee—none of the latter in the past two weeks.

Physical Examination.—Apex impulse visible and palpable in fourth and fifth interspaces inside nipple line. Heart not enlarged to percussion. At rest heart sounds are normal save for a crescendo character of the first sound at the apex; after exercise this takes on more the quality of a murmur, and yet not definitely. Slight irregularity in rhythm with deep breathing (sinus arhythmia).

Discussion.—Obviously a normal heart. The patient's army record is strong evidence in support of this opinion.

Case 12.—M. G. H. O. P. D. No. 398,528. Oct. 16, 1919. Single woman, thirty years old, bank clerk, born in Illinois.

Past History.—Chorea at ten, diphtheria at twelve, rheumatic fever at seventeen, and tonsillitis one year ago.

Present Illness.—Chief complaint is palpitation and dyspnea on exertion. Has had slight dyspnea for years, but not seriously until four months ago. Then moved to room necessitating climbing four flights of stairs. Now is exhausted and very dyspneic when this is accomplished. No orthopnea. Swelling of feet and ankles for years, but not so much of late.

Physical Examination.—Well developed and nourished. Thyroid gland shows slight bilateral enlargement. Heart: Apex impulse in fifth space about in nipple line. Sounds of good quality, rapid, and regular. Musical systolic murmur at apex, transmitted to axilla. Pulmonic second sound is greater than aortic second and is accentuated. Pulses equal, of good quality, rate 120. The murmur is increased after exercise. No presystolic murmur or thrill. No supracardiac dulness.

October 20, 1920: Referred to Cardiac Clinic.

Heart: At apex systolic murmur and snapping first sound while at rest. Weight 149 pounds. Pulse 128.

Electrocardiogram: Tachycardia, *P* wave not remarkable. No right ventricular preponderance.

℞. Sol. triple bromids, gr. xv t. i. d. p. c.

Nov. 17, 1919: Metabolism is +12 per cent. above normal. Patient is more nervous the last few days, since stopping bromids. Works fairly hard as bank clerk, but lives a quiet life otherwise. Has to climb four flights of stairs daily.

℞. Continue triple bromids. Rest.

Feb. 2, 1920: Weight 154 pounds. Pulse 104, regular. Has been working rather hard, doing extra work at the bank. Sleeps well. Symptoms are somewhat less. Has gained weight; appetite good; neck no larger.

Heart (patient in prone position): No diastolic murmur heard. Systolic whiff at the apex. Roentgen mensuration of the heart requested.

Discussion.—The above case was diagnosed as mitral stenosis at the first visit in October, 1919. In our opinion there is no evidence of mitral stenosis, but the case may be considered as "cardiac neurosis." The result of the metabolism test does not shake the latter diagnosis, as plus 12 per cent. should be interpreted as a borderline figure as regards the presence of hyperthyroidism. This patient will, of course, be followed for the possible development of definite hyperthyroidism.

SUMMARY

Of the 12 cases reported, the first 4 have definitely mitral stenosis, 2 of which have auricular fibrillation complicating it; the next 4 are doubtful, while the last 4 are not mitral stenotics, although they had been so diagnosed. One of the last group was hyperthyroidism and hypertension, another uncomplicated aortic regurgitation, a third normal with an excitable heart, and the last an example of "cardiac neurosis."

Clinic of YR

CLINIC OF DR. STANLEY COBB

MASSACHUSETTS GENERAL HOSPITAL

SPASTIC PARALYSIS IN CHILDREN[1]

IF one asks a group of medical students why spasticity is present in a lesion of the upper motor neurones the usual explanation is as follows: a cerebral lesion removes the cortical control from the "lower centers," causing increased muscle tone, exaggerated deep reflexes, and clonus. This is correct as far as it goes, but when one asks *what* "lower centers" have been freed from cortical control the answers become vague and inaccurate. It is to bring out the physiology of reflex muscle tone, and to indicate the location of the principal tonus centers that I present these 3 cases.

Case I.—Children's Medical, No. 236,355. A. J. M., age fourteen months, admitted April 26, 1920.

Family History.—Mother has marked enlargement of the thyroid and says that she is nervous. Father living and well; 4 other children living and well. No known exposure to tuberculosis or contagious disease. No history of mental diseases in the family.

Past History.—Born one month prematurely, February 15, 1919; easy delivery, no instruments used; weight 4 pounds. At birth he was very cyanotic. The doctor worked over him four hours before he came around. The cranial bones overlapped to a marked degree, and the front half of the head was narrow, as if some one had pinched it. The doctor said that he had a congenital heart lesion. When he was four hours old he had three convulsions. For the first six weeks he lay perfectly quiet,

[1] I wish to express my thanks to Dr. F. B. Talbot, on whose service Cases I and II were studied, and to Dr. Jason Mixter for allowing me to work on Case III.

with his arms bent back on his chest and his legs drawn up. They could be straightened passively, but were rather stiff. During this time he did not cry and had to be fed forcibly. His skin was dark colored and after a few weeks peeled off. There is no other history of disease except the present illness.

Feeding history: First two weeks, breast milk; then "Baby Brand" condensed milk 1 teaspoonful, water 4 ounces; feedings whenever he was hungry. The baby did not gain well and vomited frequently. At seven months he was shifted to whey 4 ounces, cane-sugar ½ teaspoonful. This made him vomit more than before, so he was changed to malted milk ½ tablespoonful, water 4 ounces, no milk. He has been on this mixture ever since, except that the amount has been increased. He has ½ teaspoonful of orange juice a day, but no solid food because of difficulty in swallowing.

Present Illness.—When the patient was six months old he began to have frequent convulsions brought on by the least disturbance, and lasting a few seconds to a minute. His arms and legs twitch, his head is drawn back and his eyes roll up. As soon as it is over he is ready to smile. This sort of thing has continued up to the present time. He also has a trick of turning blue, holding his breath, and finally coming around with a long inspiratory cry. Until he was a year old he kept his hands clenched continually and used to stick his tongue out. Now he does not do this so much. During the last four months his mother has tried to give him cereal, but each time he gags and seems unable to swallow. Liquid food causes no trouble. His appetite is good and his bowels are regular. He is apt to be rather fussy. He cannot sit up alone, and can only hold his head up for a few minutes. He knows his mother and will respond when his name is called. His sight and hearing are said to be normal. He has six teeth, the first of which appeared at six months. His anterior fontanel closed at five months.

Physical Examination.—General appearance is that of a fairly well-developed and nourished male infant of about fourteen months, not acutely nor chronicly ill, but crying lustily during the entire examination. He holds himself rigidly all of

the time. The head is drawn back slightly and his face is turned slightly to either side, usually to the left. The extremities are slightly spastic, the legs more than the arms, both sides equally. He is able to move head and extremities and is extremely irritable. At a sudden noise or pain he has a slight convulsion. The head is drawn back sharply to either side, the face usually to the right; the left arm is held upward and slightly outward, the forearm acutely flexed, the fist tightly clenched, no wrist-drop. The right arm is held close to the body, the forearm flexed at right angles to the arm, and the hand tightly clenched. The thighs are slightly flexed, the knees rotated outward. There are a few clonic convulsions and he then relaxes and begins to cry. The duration is only a few seconds. Convulsions can also be produced by introducing a throat stick or by the patient's swallowing.

Eyes: Movements and vision apparently normal. Conjunctivæ, scleræ, and corneæ negative.

Ears: Auricles are very large. Lobes are turned outward. Canals and drums normal.

Nose: Broad and rather prominent. No discharge. Septum intact.

Mouth: Lips normal. Gums normal. Six teeth. Tongue rather large, moist, not coated. Mucous membranes normal. No Koplik's spots.

Lymph-nodes: A few anterior cervical. No other enlargement.

Thorax: Normal size and shape. Rosary slight and no Harrison's groove.

Heart: Apex-beat palpable, not visible. Dulness by percussion corresponds to apex-beat. Normal rate, rhythmic and forceful. P^2 greater than A^2, not accentuated. No murmurs.

Lungs: Negative.

Abdomen: Not distended. No rigidity, spasm, or tenderness. Liver palpable $1\frac{1}{2}$ cm. below costal margin. Spleen not palpable. No hernia.

Gentalia: Foreskin long and redundant. Testicles palpable just outside of external ring.

Buttocks: Not reddened or excoriated.

Extremities: No edema or deformities. Joints movable. Hands, feet, and nails normal. Epiphysis slightly enlarged.

Measurements: Head 38.5; chest 45; abdomen 42; length 63.

Head: Smaller than normal. Lateral diameter is much decreased, especially in frontal region. The forehead is very narrow and sloping. The occiput is sloped normally, but is smaller than normal. Fontanels are closed. Suture lines not palpable. Hair is very abundant and is silky. No nits nor pediculi.

Skin: Warm, moist, and elastic. Subcutaneous fat fairly well developed. No rash or eruption.

Neurologic findings are: (1) Small skull, narrow in frontal region in lateral dimension especially; probably a "Türmschadel," with premature synostosis. (2) General convulsions. (3) Increased reflexes, with bilateral, well-sustained ankle-clonus. (4) Eye-grounds show no evidence of pressure; left disk oblong, probably astigmatic; no pathologic findings. Pupils equal, regular, and react. Chvostek's sign is absent. Neck is slightly rigid and retracted, but can be flexed on chest. Trousseau absent. Abdominal not obtained. Cremasteric not obtained. Babinski negative. Knee-jerks and ankle-jerks are exaggerated, and equal on right and left.

Electric reactions:

	Anode.	Cathode.
Right peroneal.....	5 milliamperes	1.2 milliamperes
Left peroneal.....	5 "	1.0 "
Left abdominal muscles.....	4 "	0.5 "

Above values produced contractions; less values did nothing. Not characteristic of tetany.

x-Ray: The skull sutures are not closed, but they seem rather smaller than usual in a child of this age.

Blood: Wassermann negative; Von Pirquet 24 negative, 48 negative; red blood-cells 4,944,000 per cu. m.m.; white blood-cells 14,000 per cu. m.m.; hemoglobin 80 per cent.; differential count showed polymorphonuclear cells 57 per cent., small lymphocytes 39 per cent., large mononuclears 3 per cent., eosinophils 1 per cent., red cells and platelets normal.

Urine examination showed nothing abnormal.

The stool was formed, brown, normal in constituents, was well digested and had a fecal odor.

Temperature varied between 98° and 100.2° F. (rectal), except on April 27th, when there was a sudden rise to 104° F., falling to 100° F. the next day.

Pulse ranged between 108 and 135 per minute; was 120 to 130 April 27th.

Respiration usually 30 per minute; fell on last two days to an average of 25.

Diagnosis.—Microcephalic idiot, with resulting premature synostosis of the cranial bones. Tetany was considered, but was ruled out by the absence of Chvostek's sign and of Trousseau's sign, by the character of the convulsions, the absence of laryngeal spasm, and the presence of abnormal reflexes. The normal electric reactions substantiated this opinion.

Case II.—Children's Medical, No. 235,922. L. G. T., age four years, admitted March 31, 1920.

Family History.—Father living and well so far as is known. Mother had tuberculosis as a child and was in an English sanatorium for several years. At present she is in poor general condition, but when she was examined in the Female Medical Out-Patient Department a month ago she was told that she did not have any active process now. One sister, underweight, gets sick easily, but has no definite disease. No miscarriages.

Past History.—Born at full term, normal delivery. The mother was in bed seven weeks with pneumonia while pregnant. He was bottle fed—Mellin's Food and milk, formula not known. When he was teething he had convulsions and vomited repeatedly. His appetite is poor and his bowels fairly regular. No contagious diseases. He sat up alone at one year, talked at three years, and walked at two years.

Present Illness.—Patient was brought into the Out-Patient Department because he was underweight, backward, and because he has always dragged his right leg when he walks. He runs very little, falls often, and has to be carried up and down stairs. He does not talk readily, is slow, unresponsive, and seems very

tired at night. He plays with other children. Since early in January he has wet the bed at night. In the Out-Patient Department the throat department said that his tonsils were moderately large, and suggested operation if fixing his teeth did not improve his condition. The dentist reported that his cavity was superficial and would not account for the cervical glands. The orthopedic department said that "the gait was not significant" and that it was "not an orthopedic problem." The nutrition clinic sent him to the Fathers' and Mothers' Club for five weeks, where he was able to get fresh air, rest periods and lunches, and went to bed at 6 P. M. He gained only ½ pound, however, and he is sent into the house for further study.

Physical Examination.—The general appearance is that of a well-developed and fairly well-nourished male child of about four years, not acutely ill. The child does not impress one as being mentally backward. He seems a little bashful, but understands anything that is said to him. He walks with a peculiar gait; the steps are short and he raises his feet rather high. The right foot is slightly more everted than the left. There is a moderate degree of flat-foot. He is able to rise from the floor normally.

Skin: Warm, moist, and elastic. Subcutaneous fat fairly well developed. No rash or eruption.

Head: Normal size and shape. Fontanels closed. No nits nor pediculi noted.

Eyes: Apparently sees normally with both eyes. Movements normal. No discharge. Conjunctivæ not inflamed or anemic. Scleræ and corneæ normal.

Ears: Normal size and shape. Canals normal. Drums normal.

Nose: Normal size and shape. No discharge. Septum intact.

Mouth: Gums normal. Teeth 20; two cavities are superficial. Mucous membranes normal color. No Koplik's spots.

Throat: Tonsils are slightly enlarged, surface rather scarred and are bound to the pillars. He is not a mouth-breather. He talks very peculiarly; tones have a definite nasal twang, but with apparent change when nose is held.

Lymph-nodes: Few anterior cervical glands. No other glandular enlargement.

Thorax: Normal size and shape, no beading or Harrison's groove.

Heart: Apex-beat palpable and corresponds to heart borders. Normal rate forceful and regular. P^2 equals A^2. No murmurs.

Lungs: Clear.

Abdomen: The level is that of the thorax. No rigidity, spasm, or tenderness. Liver and spleen not palpable. No hernia.

Genitals: Prepuce can be retracted. Testicles descended.

Buttocks: Normal.

Extremities: No edema. No deformities of extremities. Joints movable.

Measurements: Head 48; chest 54; abdomen 54; length 106 cm.; weight 15.7 kilograms.

Neurologic: Cranial nerves negative (fundi not well seen). Motor system shows: (1) Spasticity of both legs, right slightly greater than left; the gait is not typical; the child seems to have learned to pick up his knees in order to overcome the tendency of the toes to scrape. No muscular weakness, no atrophy. (2) Reflexes: knee-jerks and ankle-jerks exaggerated and equal on right and left; patellar reflexes active and equal, right and left; ankle-clonus bilateral, well sustained, right slightly greater than left; Babinski bilateral, right greater than left. Arm reflexes are exaggerated and equal. Sensory examination not done. Endocrin system negative. Pupils equal, regular, and react to light. Mentally somewhat retarded, but this cannot be definitely decided.

Temperature between 97° and 100° F. (rectal).

Pulse ranged from 75 to 105 per minute.

Respiration ranged from 20 to 28 per minute.

Urine: Straw color, acid reaction, no albumin, no sugar, sediment negative (three examinations).

Blood: Red blood-cells 4,168,000; white blood-cell count shows 12,600; hemoglobin 70; differential count shows polymorphonuclear cells 72, mononuclears 28, red cells and platelets

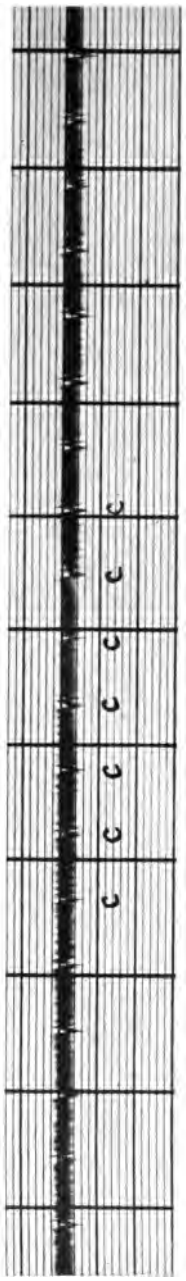

Fig. 47.—Electromyogram of ankle-clonus, showing a rate of about 8½ contractions per second. This record was obtained by connecting the patient with a string galvanometer, such as is used for electrocardiography; the registering electrode is placed over the contracting muscle (in this case the gastrocnemius) and the indifferent electrode over the inner side of the thigh. The muscular contractions of the clonus then send action-currents through the galvanometer, which are photographically recorded as the large waves shown in the figure (*c*, *c*, etc.). The vertical lines are chronographic, the interval between each pair being one-fifth of a second.

normal. Wassermann negative. Von Pirquet, 24 strongly positive, 48 strongly positive.

Stool: Formed, bran color, negative except for vegetable fibers.

Lumbar puncture: About 6 c.c. of crystal clear fluid not under increased pressure. Cell count 4 per cu. mm. Smear negative for organisms. Cells endothelial. Culture negative. Globulin negative. Wassermann negative. Colloidal gold negative. Pellicle negative.

x-Ray: Diaphragms are rather flat, costophrenic angles clear. Respiratory movements normal. Hilus shadow increased on both sides. There is a good deal of fine mottling present along lung markings. Some of these shadows are unusually dense, suggesting calcification. Appearance is that of a glandular, possibly peribronchial, tuberculosis.

Basal metabolism: Averages 34.4 calories per hour, or 1120 calories in twenty-four hours per square meter.

Electromyographic study of ankle-clonus: Shows the average rate of the clonus to be 8.5 contractions per second; the periods between the con-

tractions are regular, and the time from the beginning of one contraction to the beginning of the next measures almost invariably 0.12 second. Bewteen the larger waves (Fig. 47), each group of which indicates a clonic contraction of the gastrocnemius, are fine waves due probably to the action currents accompanying a slight voluntary push exerted by the child's leg.

Diagnosis.—The case is probably one of spastic paralysis following a birth injury. The prognosis is good. The paralysis is only slight and will probably clear up by the time adult life is reached. The fact that the spasticity is somewhat more marked on the right than on the left, with clonus greater on the right than on the left, suggests a birth injury more than encephalitis. The electromyogram gives definite evidence as to the organic etiology of the clonus.

Treatment.—Present treatment should be massage and exercises. It is not thought that the tuberculosis is at all active.

Case III.—W. S. No. 235,337. R. B. P., age six and a half years, admitted February 25, 1920.

Family History.—Not remarkable.

Past History.—Born three weeks after full term. He has had no illnesses except measles at two and chickenpox at four and a half years of age. His development has been unusually precocious, teeth appearing early, walking and talking exceptionally early, and his weight was 25 pounds at five months. This precociousness has continued and his mother says he seems older than other children of his age.

Present Illness.—In November, 1916 he began to show lassitude and complained of fatigue. After three weeks of this, on December 14th, he vomited, complained of headache, and then went into a state of generalized rigidity which lasted for half an hour and was followed by a general convulsion and vomiting. He was then rigid again for three hours and stuporous. Lumbar puncture by his attending physician relieved the symptoms, and he had a slow, drowsy return to consciousness over a period of twenty-four hours. A diagnosis of brain tumor was

made and he was sent to the Peter Bent Brigham Hospital (No. 11,228) for study on Dr. Cushing's service. Here the following positive findings were observed: A child of three, very muscular and rather overdeveloped for his age. His responses to questions are prompt and more intelligent than might be expected. There is a limitation of the motion of the eyes upward, and slight nystagmus on looking to either right or left. The finger-to-nose test elicits dysmetria, and there is an ataxic gait. The reflexes are sluggish. Both fundi show choked disks of 2 dipoters. *x*-Ray of the skull shows a shadow in the pineal region and indications of hydrocephalus.

The patient improved on the ward, so operation was deferred and the child sent home for further observation. The diagnosis of "pineal tumor" was tentatively made.

Shortly after returning home he had visual disturbances, vomited occasionally, and in April had a severe headache, so he was taken to another hospital and there operated on for cerebellar tumor. The notes follow: Operation May 4, 1917. Suboccipital decompression. Dura very tense over cerebellum. Ventricle tapped, relieving tension. Dura incised from side to side across midline. Two vertical incisions over both cerebellar lobes, but no definite tumor felt. Wound closed; dura stitched; periosteal and muscular flap stitched and skin closed. He made good recovery and for about two weeks improved in co-ordination; no headaches or vomiting. May 18, 1917 swelling and bulging of right side of cerebellum. May 27th vomited twice; inco-ordination; marked headache; more tension on right side of cerebellum. June 2d and 6th lumbar puncture with increased pressure and temporary relief following. June 12th tension same, but no further headache or vomiting; eyesight good; reflexes normal, unsteady when sitting alone; discharged to home. Since then he has not been able to walk. His left leg and later on his left arm troubled him; he could not control them well. His vision has grown worse, especially the last three months. The mother thinks that he has grown worse in the last three months, although he has not had so many severe headaches. His speech is slower than usual, but mentally he is alert.

Physical Examination.—Well-developed and well-nourished boy of six lying quietly in bed.

Skin: Clear and soft; mucous membranes good color.

Head: Large. Over occipital area is a large bulging protuberance.

Eyes: Scleræ clear. Lateral nystagmus—the eyes shifting to right and snapping back to the left. Pupils both fairly well dilated; right larger than left; neither reacts to light; at a distance of 1 foot the child can rarely tell how many fingers are put up.

Ears and nose: Slight nasal discharge.

Mouth: Tongue protruded to the left, clean, no coat.

Facial muscles: Some drooping to the left; could not get him to smile.

Neck: Negative.

Chest: Well developed; expansion, right equals left.

Lungs: Clear and resonant throughout.

Heart: Normal size and position; sounds regular, rapid; no thrills or murmurs.

Spine: Negative; no costovertebral tenderness.

Abdomen: Level, soft, and tympanitic; no spasm, masses, or tenderness; no organs felt.

Genitals: Neither testicle felt in scrotum.

Extremities: Right arm moves with some difficulty of coordination and coarse tremor. This is more marked in the left arm and hand. The grip of the fingers of the left hand is much weaker than the right. The left hand is held with fingers flexed most of the time.

Legs: Both feet are held in equinus—especially the left, with the big toes extended. Both legs are more or less rigid.

Neurologic: There is secondary optic atrophy in both eyes. The other positive neurologic findings are inability to move the eyes to the left, weakness of the left facial muscles, paralysis of the left arm with paresis and ataxia of the right arm, and paresis of the legs. The striking thing is the extensor position of the legs with increase of muscular tone. Especially interesting is the flexion reflex from stimulation of the right plantar, and the

crossed extension reflex in the right leg from stimulating the left plantar. The arms are also stiff at shoulder and elbow, but loose at the wrist; they are usually held in partial flexion at the elbow. The reflexes show normal knee- and ankle-jerks; no Kernig; no clonus obtained; Babinski reflex is questionable, as the toes are held in the extended position; on tapping the left

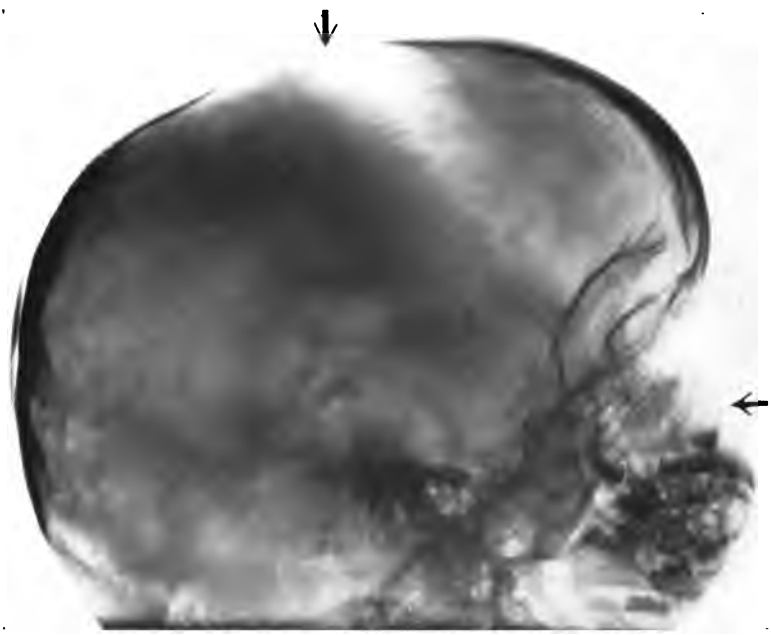

Fig. 48.—*x*-Ray of skull of Case III, lateral view, showing shadow in region of pineal body. (This can be located by following out lines in the directions indicated by the two arrows, the tumor shadow being where the lines would cross.

knee the hamstrings of the right leg contract. The child is incontinent of urine.

Eye consultation, February 27th: Disks well outlined; very pale; vessels of good size and tortuous; retinæ normal. Secondary optic atrophy.

Temperature: Between 98° and 99.8° F. until just before death, when it rose to 105.8° F.

Pulse: 125 on admission; while on ward averaged 90; reached 170 before death.

Respiration: 15 to 25; 48 before death.

Urine: Straw colored, clear, acid reaction, no albumin, no sugar, no diacetic acid; the sediment shows uric acid crystals in excess, no red cells, pus cells, or casts.

x-Ray: Skull unusually large and thin. It presents a mottled appearance suggestive of intracranial pressure. Sutures wide in

Fig. 49.—Photograph of sagittal section of the brain of Case III, showing large teratoma of the pineal body, causing great pressure on the midbrain and displacement of the cerebellum and pons. The dilated ventricles indicate the extent of the hydrocephalus.

upper portion of skull. Just above mastoid process in midportion of skull is a calcified area which has a horseshoe shape. This mass is apparently in the brain, and coincides with the position of the pineal body (Fig. 48).

Operation (March 5th).—Ether anesthesia. Ventricular puncture and injection of air; 700 c.c. fluid withdrawn through puncture in frontal region in small amounts (20 c.c. after the

first 200 c.c.) and air injected to replace same for ventriculography.

x-Ray (March 5th): Plates taken of skull after injection of ventricle with air show the outline of a pedunculated mass about 1¼ inches in diameter rising from the posterior part of the floor of left lateral ventricle. The dense shadow seen in previous plates is apparently in the center of this mass. The ventricles are extremely large and the amount of brain substance very much reduced. The appearance is that of extreme internal hydrocephalus, probably due to a tumor rising near the aqueduct of Sylvius. The fact that there is calcification in the tumor would suggest that it is of epithelial origin.

On March 6th the child died, and autopsy showed a pineal teratoma (Fig. 49) displacing the midbrain, shutting off the aqueduct, and causing extreme internal hydrocephalus.

Diagnosis.—The history taken at the Brigham Hospital, which brings out the precocious development of the child, first drew attention to the pineal body as the probable seat of the lesion. When symptoms of pressure on the midbrain, of "decerebration," and of internal hydrocephalus appeared the diagnosis was more probable, and the *x*-rays finally made it certain that a tumor of the pineal gland existed, probably an epithelial growth.

These 3 cases all show hypertonicity of the limb muscles with hyperactive tendon reflexes and clonus. The diagnoses indicate that in all 3 the cerebral cortex is not properly functioning: in the microcephalic case, because of its lack of development; in the birth hemorrhage because of local destruction; and in the pineal tumor on account of the pressure of the growth on the motor tracts as they pass downward in the cerebral peduncles and into the midbrain. This indicates the correctness of the theory that spasticity develops when "cortical control" is removed. But clinical data has given us little evidence as to the location of the centers which become hyperactive after the removal of this higher control. By turning to the experimental work of physiologists, especially Sherrington, we can find important analogies in the reactions of the "decerebrate" animal.

It has been demonstrated[1] that when the cerebrospinal axis of an animal is transected through the anterior part of the midbrain at the level of the superior colliculi, and (Fig. 50, *T*) above the red nucleus, that this animal goes into a state of tonic muscular contraction known as *decerebrate rigidity*. This tonic contraction is confined to the extensor muscles of the limbs and the elevators of the neck, tail, and jaw—in short, to those muscles that hold the animal in an upright posture and thus counteract the steady force of gravity. In other words, a complicated mechanism that holds the animal in the *reflex posture of standing* gains control of the musculature. We say *reflex* advisedly, because it has been shown by Sherrington and others that the rigidity disappears when the afferent impulses are cut off by section of the posterior roots or of the lateral columns of the cord. Thus it is proved that the ultimate source of the stimuli that set up this rigidity lies in the peripheral sense organs, especially those deep sense organs that subserve muscle, tendon, and joint sensation. The phenomenon is therefore properly called a *postural reflex*. More recent investigators[2,3] have located the main reflex center for the standing posture in the red nucleus of the midbrain. This was accomplished by making successive transections of the brain stem until the reflex rigidity disappeared. Subsequent microscopic examinations of the serial sections of brain removed then showed that injury to the red nucleus caused loss of all the steady rigidity. A certain amount of postural reflex, however, remained until the vestibular nuclei were injured. This accords with the theory of other workers, notably Ewald,[4] who believe that muscle tone has an important center in the otic labyrinth.

From the clinical standpoint it has been shown by Wilson[5] and Hunt[6] that the corpus striatum (*i. e.*, the caudate and lenticular nuclei) has an important control over muscle tone and simple motor associations such as those necessary for locomotion. Pathologic processes in these nuclei cause abnormalities of tonus, for example, the muscular rigidities seen in Wilson's disease and paralysis agitans. Walshe,[7] in a recent paper on spasticity,[7] has ably summed up the subject, and says that the ex-

tended type of spastic paralysis is identical with the "decerebrate rigidity" described by Sherrington in animals, that the spastic element is the same phenomenon as the tonic extensor rigidity seen in these animal preparations, and possesses similarly a mesencephalospinal reflex arc.

We have in these 3 cases symptoms of uncontrolled tonic activity with resulting spasticity, hyperactive reflexes, and clonus. In Case I the lesion is in all probability a bilateral aplasia of the cerebral hemisphere (Fig. 50, *A*) with the physio-

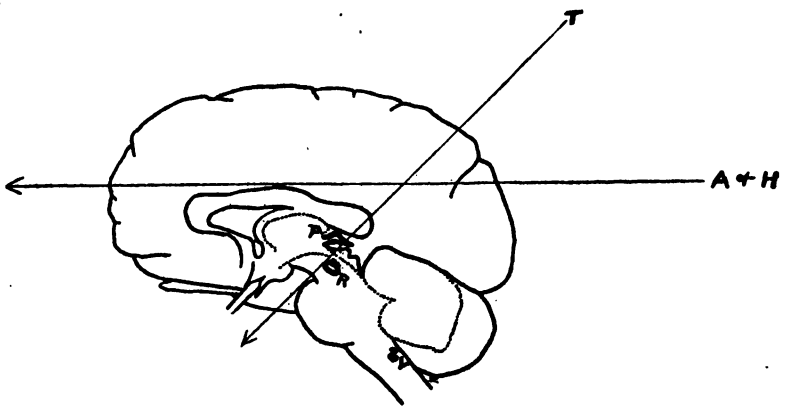

Fig. 50.—Diagram of sagittal section of a normal brain: *P*, Pineal body; *R*, red nucleus; *V*, vestibular nuclei. A transection of the brain along the line *A* and *H*, removing the cortex, gives the symptomatology of "decortication." Transection along the line *T* passes through the midbrain and causes the symptoms of "decerebration."

logic results that would follow decortication in an animal. Case II represents an extensive lesion of the cerebral cortex (Fig. 50, *H*), probably a hemorrhage from birth trauma causing more damage to the left hemisphere than to the right, with symptoms similar to Case I, but with more increase of muscle tone on the right side of the body than on the left. Case III has a lower lesion where the converging projection fibers from the motor cortex are concentrated in the peduncles and midbrain so that pressure by the pineal tumor brought about, in fact, a true decerebration (Fig. 50, *T*), and the symptomatology described

in the physical examination bears a notable resemblance to the phenomena of decerebrate rigidity described by the physiologists.

The cases differ in voluntary power and degree of spasticity. This can also be explained by a comparison with physiologic experiments, for if the cortex of an animal is removed, leaving intact the corpus striatum and other nuclei of the basal ganglia, the power to associate movements for walking, climbing, and the like is conserved, and the animal is still able to carry out all of the fundamental acts of life. In addition, there is an increase in muscle tone, especially in the extensor muscles which keep the standing posture. This in a crude way describes Cases I and II, although in neither of them is there reason to suppose that the whole motor cortex is lacking. So the symptoms are not so severe as those described for the animal. But physiologically speaking these can be called cases of partial decortication. In Case III, however, there is no ability to walk, the arms are asynergic, and tonus is greatly increased in the muscle groups necessary to maintain the reflex standing posture, thus showing that the red nucleus is functioning. The loss of simple motor associations, such as the movements of walking, indicates that the basal ganglia are cut off, so the physiologic diagnosis of *decerebration* can be made, and the lesion located somewhere in the motor tract below the lenticular nucleus and above the red nucleus.

It is well to consider the subject of motor power and tonus from another point of view. We have been speaking of the central nervous system and its effect on the muscular system in general. Now let us take a muscle as the starting-point and analyze the effects that play upon it:

The muscle is innervated only by the peripheral nerves, the motor fibers of which all arise from the anterior horn cells of the spinal cord. Any motor impulse, therefore, that reaches the muscle must travel along this path, and since the whole muscle acts as a unit in contracting, it may be taken for granted that the simultaneous impulses coming down each of the many nerve-fibers of the peripheral nerve are similar one to another. In our

diagram (Fig. 51) we can then draw the peripheral motor nerve (F. C. P.) from the anterior horn cell to the muscle as if it were a single fiber. But there are many impulses that play on the anterior horn cell and affect the impulse that goes to the

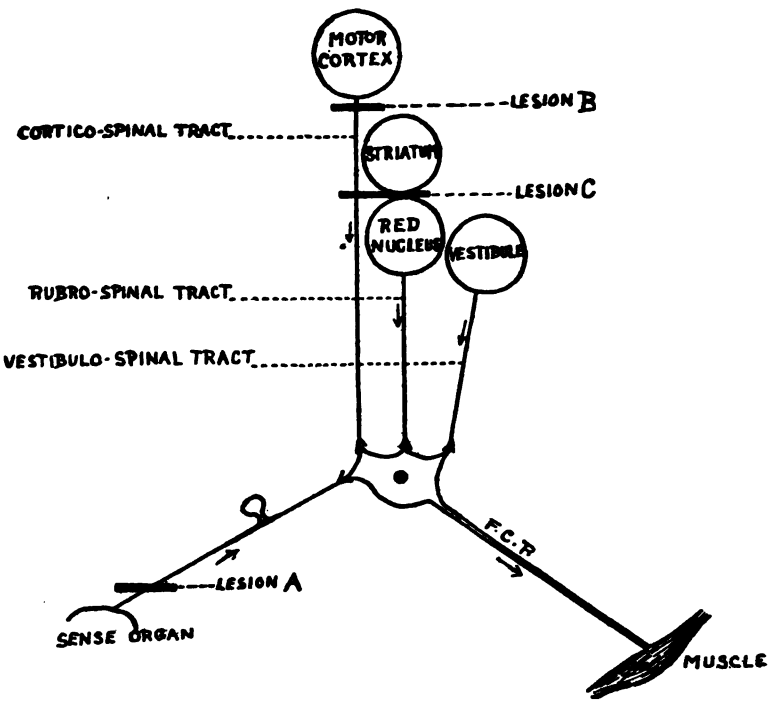

Fig. 51.—Diagram of the motor pathways (modified from Wilson). Impulses from the peripheral sense organs, from the motor cortex, the striatum, red nucleus and vestibule, are indicated as passing down their respective tracts to concentrate on the anterior horn cell. Thus the series of impulses which travel down the final common path of the peripheral nerve, to the skeletal muscle, is a composite and modified energy, according to the strength of the innervation from each of the great centers. (For examples and descriptions of lesions, see text.)

muscle, so the motor nerve-fiber is not only a final path for motor impulses, but it is the final *common* path down which all motor impulses must travel to reach the muscle, no matter what their source.[8]

With this conception of a *final common path* for the motor

impulses clearly in mind, let us sketch the various sources of motor energy which play on the anterior horn cell.[9] In the first place, impulses received in the peripheral sense organs reflexly affect muscle tone through short reflex arcs across the spinal cord, without bringing into play any of the brain centers at all. This simple reflex arc, though crude in its function, is all important, for if it is cut (lesion A, Fig. 51), as in section of the posterior roots, no reflex muscle tone remains, and the higher centers, acting, as it were, blindly from above, have but a toneless ataxic muscle to play upon.

The second great source of motor innervation is from the motor cortex via the corticospinal path, and with a lesion in this tract at a level higher than the striatum (lesion B, Fig. 51) we impair or destroy the power of isolated voluntary movements. We say "isolated movement" because in injury to the corticospinal tract alone it is these highly specialized, single and purposeful movements that are lost, not the gross associated movements of locomotion and bodily station. This condition is illustrated by the partial lesions of the cortex in Cases I and II.

The lower lesion caused by the pineal tumor in Case III, however, cuts off the effects of the striatum as well as those of the cortex (lesion C, Fig. 51), so the uncomplicated impulses from the red nucleus and vestibular nuclei play on the anterior horn cell, and give the phenomena of decerebrate rididity. As described above, physiologic experiments have shown the effects of the red nuclei, and then the vestibular nuclei. This leaves the isolated spinal cord acting as a reflex center, and explains the great loss of muscle tone seen after cord transection, as well as the possibility of quite extensive and complicated reflex action.

In recapitulation, then, we can say that fundamentally the afferent impulses pouring into the central nervous system set up in the muscles that slight constant contraction we call *tonus*. Some of these impulses cross directly to the anterior horn cells via the simple spinal reflex arc, while others travel up to the brain and give rise to the energy which emanates from the great tonus centers in the nuclei of the striatum, in the red nucleus, and in the vestibular nuclei. On this whole mechanism the cere-

bellum has a great co-ordinating influence, and also an important though not well understood, tonic function. Lastly, the motor cortex of the cerebrum controls all these lower mechanisms through its power of initiating the isolated voluntary movements, which take temporary precedence over all other forms of motor activity, but are usually superimposed on these tonic postural reflexes, and do not truly inhibit them.

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CLINIC OF DR. MAYNARD LADD

CHILDREN'S DEPARTMENT, BOSTON DISPENSARY

VOMITING AS A SYMPTOM IN CHILDREN

VOMITING in children may be due to the simplest of causes, or may be a symptom of very serious significance and due to many different conditions. Its meaning should always be sought, and its occurrence never passed over lightly.

Physiologically the stomach of the newborn is at best an imperfect reservoir, with little or no development of the cardia and a pylorus that exercises very little of the function seen in later months. Consequently, overdistention from too generous feedings or sudden changes in the position of the infant result in an overflow—simple regurgitation.

The stomach contents pass quickly into the duodenum as long as the food remains in a liquid form, and gastric digestion in early life is relatively unimportant in comparison with duodenal digestion. The soft flocculent curds of breast milk offer little obstruction to direct passage through the pylorus. In general, one may say that any measures taken to lessen the formation of large casein curds facilitates normal gastric peristalsis, and contributes to the solution of the problem of successful feeding.

The growth of the stomach is rapid in the early months, and the quantity allowed at each feeding must be regulated in proportion. The breast-fed child takes a much larger quantity at a feeding, if the supply permits, than it is wise to give in substitute feeding, hence the wisdom of fewer feedings and longer intervals in breast-fed babies. The same large amounts of a substitute feeding are retained a longer time, the emptying time of the stomach is prolonged, opportunities for fermentation, with gas formation, occur, and eructations with forcible vomiting takes

place. With such symptoms pain is manifest and we begin to see the picture of *gastric colic from indigestion.* Uncorrected, these conditions favor the development of dilatation of the stomach, still more retardation of normal peristalsis, with a state of marked discomfort and indigestion.

There is no one element in the food that is responsible for this condition. It depends upon imperfect adaptation of cows' milk and inattention to the various factors which are concerned in successful feeding; that is to say, the strength of the food as a whole, the proper relations of the ingredients to each other, the appropriate quantities at each feedings, the length of time taken at a feeding, the proper temperature of the milk, etc. The correction of regurgitation or simple vomiting from indigestion, therefore, involves an appreciation of all the varied factors in infant feeding, which is not within the scope of the present clinic.

In older babies, badly fed for prolonged intervals, one can recognize types of *indigestion more or less closely associated with some particular food element.* A careful analysis of the previous feedings, with an estimation of the food values, generally points the way to a diagnosis of the cause, and aids one in making the necessary correction. Any element in excess of the normal food requirements may be at fault; fat is the chief offender, proteins next, and carbohydrates least of the three. Percentage feeding teaches us the principles of food analysis by estimation, and greatly aids us in coming to a conclusion as to the faults of the previous feedings and the best and quickest way to correct them.

One point should be kept clearly in mind. Vomiting due to food indiscretions is quickly relieved if the condition is properly diagnosed and the feedings intelligently and scientifically directed. Vomiting which is not relieved, or which by the primary analysis of the case cannot be reasonably explained on a basis of improper feeding, must be looked upon with suspicion, and more serious conditions considered.

For instance, an infant, in the early days or weeks of life, from the very beginning and in spite of perfectly rational feeding, begins to vomit violently, that is, with a force and explosive

character quite unlike regurgitation or vomiting from indigestion. The vomiting occurs usually at the end of the feeding without warning and the food is expelled a foot or more from the child. Most of the feeding comes up, but the baby does not look sick, cries vigorously from hunger, and is quite ready to begin all over again on another bottle, but with the same result. How many times one has seen such a case, passed over for weeks, innumerable changes in the food made, while the child rapidly emaciates and looks and acts like a starved animal, vigorous and protesting in the early stages, but gradually losing in strength and weight, until in its last stages we have a picture of a prostrated infant suffering from inanition and intoxication, and often dying. This distressing situation might easily be avoided and the condition of *pyloric stenosis* diagnosed in the very early stages when operation promises every hope of a cure. The physician starts with the hypothesis of food indigestion, never examines the baby for visible peristalsis and tumor, and often fails completely to appreciate the significance of the symptom of vomiting until the child's chances to live have passed.

Easily confused with such cases are those which we classify as *pylorospasm*, a condition in which so far as the vomiting is concerned may readily make us suspect a stenosis, although the vomiting is usually more in the nature of regurgitation, is less violent, but often as resistant to changes in the food. Visible peristalsis is not seen in these cases unless some degree of stenosis exists, and a pyloric tumor cannot be felt. A considerable amount of food passes into the intestines in spite of the spasm, and the loss of weight is less in amount and less rapid than in pyloric stenosis; often sufficient food is retained to maintain or nearly maintain the body weight. We do not know all the factors on which pylorospasm depends, excluding the cases associated with slight stenosis, nor can we say what type of feeding will best overcome the condition, except that, whenever possible, breast milk should be used, and if that is impossible, modified milk with low fat percentages, relatively high sugars, and proteins either predigested (peptonized) or so changed as to prevent large curd formation in the stomach. Curds are the

chief factor in exciting spasm when the tendency exists. Possibly there is some sensitization of the stomach to the protein, but there is no scientific data for such a hypothesis, although some clinical evidence is in favor of the theory. Whatever the cause, the tendency to vomit gradually wears off, providing one can adapt the food to the child in such a way as to promote normal or approximately normal weight development.

Cases of pyloric stenosis are easily diagnosed if the physician is alert to the significance of the symptom of vomiting. Operation is indicated if the child cannot be made to gain in weight in spite of it. Pylorospasm not associated with stenosis is not benefited by operation. There is no class of cases more difficult to feed or that will tax one's resources to a greater extent. Unfortunately, the food that will bring about a satisfactory weight development in one case, fails completely in another, but with persistence and with exact attention to details of feeding the prognosis is good. The difficulty of keeping the confidence of the parents through the weary weeks of study and adaptation of the milk is very great.

When a child has been progressing satisfactorily in its feeding for weeks or months and suddenly begins to vomit, without any known change in the food or indiscretion on the part of the mother, one must look upon the symptom as potentially serious. Such vomiting may, of course, be due to some deterioration or infection of the milk; if so, catharsis, withdrawal of milk, simple gruels, or water or soda solution generally result in relief of the symptoms. Usually we must look for the development of other conditions of which the vomiting represents the red flag of danger. We are all aware, for instance, that the onset of *any acute disease* may be ushered in with vomiting, especially scarlet fever and acute intestinal infections, which within a few hours are manifest by characteristic stools.

Vomiting with high fever may be due to many causes, which are usually quickly localized by characteristic additional symptoms. There are, however, two conditions very frequently overlooked because of faulty or insufficient investigation. These are *acute otitis media* and *acute pyelitis* or *pyelonephritis*. Un-

fortunately, the average physician in family practice does not, as a matter of routine, make use of the auroscope. He also is careless in securing a specimen of urine, considering it difficult to obtain, whereas it is the simplest thing in the world to do. All that is needed is a good-sized test-tube, held in place with adhesive plaster and allowed to point downward, the child's body being placed on a pillow, and the legs, immobilized by diapers, holding the tube in a fixed position. The urine is readily collected.

Now both otitis media and pyelitis may attain a considerable degree of development without any localizing signs or symptoms to make one suspect their presence. The high fever, marked prostration, pain which is not localized may lead one to suspect pneumonia, influenza, tonsillitis, and various other conditions. Vomiting is a common symptom in these affections. It is often attributed to indigestion, but indigestion is rarely a cause of vomiting when fever is prominent unless the stools show evidence of fermentation (sour, foul, slimy, green) or of infection (blood, mucus, pus). No case of vomiting with fever in an infant should be allowed to pass without an examination, at the very onset of symptoms, of the ears and of the urine.

Sudden vomiting, without fever or with very slight elevation of temperature, in a child previously well, is often due to an *acute abdominal condition*. In considering possible explanations of the symptom, one notes that along with the vomiting the child has a facial expression indicative of pain or anxiety or prostration, quite different from that produced by the pallor and discomfort of an indigestion. The absence of any considerable degree of fever must not lead us to overlook an acute intestinal condition in which obstruction plays an important part. It is a help in diagnosis to know that acute appendicitis is excessively rare under one year of age. I have never seen a case. It occurs less rarely in the second year and after that period is always a possible cause. Having associated the symptoms of vomiting with some acute abdominal condition, we are much more liable to be dealing with a strangulated hernia, an intussusception, or a volvulus than with a diseased appendix.

I do not intend to go into the differential diagnosis of acute intestinal obstruction, but I would simply emphasize the importance of bearing in mind the close connection of vomiting with serious abdominal conditions, and the fatal delays that often occur as a result of attributing the symptom of vomiting to some acute food disturbance, when nothing of the sort exists except as a secondary condition. Acute severe vomiting, especially when associated with pain, should be immediately and carefully investigated. Too many doses of castor oil are ordered at the first, and the examination of the abdomen left to a later visit. As a result, strangulated herniæ and intussusceptions are often allowed to exist for thirty-six or forty-eight hours before the correct diagnosis is made, and it is needless to emphasize how such delays jeopardize the chances of the child.

There is another interesting condition, of which vomiting is the main symptom, which we see in children who are of a distinctly *nervous type*. It has a tendency to recur at irregular intervals, weeks or months elapsing between attacks. The child is very likely to wake up from a sound sleep violently nauseated. If the stomach is full, undigested food is expelled, but without relief of symptoms, which one expects to follow in simple dyspepsia. Subsequently the child continues to retch. There is considerable thirst, but even water is expelled and the simplest kinds of food as well. A condition of intestinal stasis develops, with obstinate constipation, and any attempt to move the bowels from above generally makes the vomiting worse. A sweetish odor on the breath may be detected and the urine, which is passed freely, has the same characteristic odor, and on examination shows acetonuria. The period of vomiting, which is very frequent and quite independent of feeding, may last for twelve hours or several days in severe cases, but is rarely fatal. Parents are inclined to call the attacks "bilious" because bile appears in the vomitus when the retching is severe. There is, however, no relation between the condition and the function of the liver. It is due, so far as we know, to some obscure disturbance of metabolism within the tissues. We must realize the close interrelation of fat and carbohydrate metabolism. Too

much of the former or a deficiency of the latter food elements are the chief contributing factors in the disturbed state of metabolism which the attack indicates. The history of previous attacks may put us on the track of the right diagnosis and relieve our minds of the anxiety which the appearance of the child arouses. Of course there is always a first attack in these cases of *"cyclic"* or *"persistent" vomiting*, and, on the other hand, a child who has been subject to periodic attacks may be the victim of an acute intestinal obstruction which the symptoms suggest. So great care must be exercised in the differentiation, but if from the previous history and from the characteristic attacks, and from the acetonuria at the very onset of symptoms, we form a correct diagnosis, we will not make the mistake of treating the case as if it were primarily gastric indigestion with constipation, nor will we make the fatal mistake of doing an exploratory laparotomy on a child who is free from obstruction and who is the worst of surgical risks because of the condition of acetonuria.

True *acidosis* does occur in infants and young children with which the condition of cyclic vomiting must not be confused. Acidosis rarely develops even in protracted cases of cyclic vomiting. When it does occur it is pratically always preceded by some primary exciting cause which our history and examination should determine. Nephritis, diabetes, severe intestinal infections, or some severe acute respiratory diseases may have as their late and final manifestations the typical picture of acidosis, the red lips, extreme pallor of the skin, persistent vomiting and air-hunger, with diminished carbon dioxid tension of the alveolar air, and increased tolerance to bicarbonate of soda. These cases are usually fatal, but the cyclic vomiting which we have in mind, which is recurrent and due to obscure disturbances of metabolism, fortunately has an entirely different prognosis.

Diseases of the *central nervous system* are generally ushered in by vomiting. The localizing symptoms at first may be so slight as to be overlooked, except in severe acute inflammatory conditions, such as meningitis, encephalitis, etc. The more slowly developing diseases, such as tuberculous meningitis,

internal hydrocephalus, and brain tumors, are not easily diagnosed in their incipient stages. The vomiting which occurs in these conditions is usually violent, but is not frequently repeated. Several days may elapse between attacks, and we are likely to make a diagnosis of vomiting from gastric indigestion until other symptoms of cerebral irritation point to the true cause of the vomiting.

There are a variety of minor conditions which produce vomiting which we must keep in the field of possible causes until the true nature of the case is determined. Such vomiting is purely a *reflex* and has many origins. Intestinal parasites, swollen, tender gums from difficult dentition, foreign bodies, eyestrain, fright, nervous excitement, fatigue, pharyngeal irritation from hypertrophied tonsils, elongated uvula, rectal disease, and many other conditons will, upon consideration, suggest themselves as occasional causes of vomiting. Very rarely one runs across a case of *rumination*, a condition in which nursing babies, somewhat after the fashion of a cow who chews her cud, deliberately bring up portions of a meal, to be swallowed again, until finally the whole gastric contents may be expelled. Again, we have all seen the child who continually forces its hands far back into the mouth, until by reflex irritation of the pharynx or larynx vomiting is excited. Severe *paroxysmal coughing spells*, such as we see in pertussis, peribronchitis, and enlarged mediastinal glands, or localized pharyngeal abscess, often terminate in a severe attack of vomiting, but the cause is so obvious that one seldom misinterprets it.

Reflex irritation from any of these causes in neurotic children may lead to a state of "*habit vomiting*," but such a diagnosis, hastily made, has its dangers, in that we may not seek for some of the more serious causes of vomiting to which we have alluded.

This review of the significance of vomiting in young children is cursory and, doubtless, incomplete. It will have served its purpose if it will encourage the physician to pay more attention to the symptom when it occurs in young children, and to look for associated symptoms which may result in earlier and more careful

differentiation in diagnosis. "Indigestion" covers a multitude of mistaken diagnoses in early stages of many different conditions, and unless we have in the back of our heads a pretty clear conception of vomiting in its various manifestations and with its varied associated symptoms indicating the nature of the primary cause, we are liable to lose valuable time in making a correct diagnosis and in instituting the appropriate treatment.

FROM THE MEDICAL CLINIC OF THE BOSTON CITY HOSPITAL

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By Edward H. Nichols, M. D.

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CLINIC OF DR. EDWARD H. NICHOLS

BOSTON CITY HOSPITAL

SUGGESTIONS REGARDING THE EARLY DIAGNOSIS OF ACUTE APPENDICITIS

THE classic case of appendicitis is almost universally recognized by the profession at large and everybody accepts operation as immediately indicated. The classic case commences with acute abdominal pain, which may begin as general "cramps" in any part of the abdomen, quickly localizing in the right lower quadrant. This pain may be intense. Associated with the abdominal pain are symptoms of obstruction, that is, constipation or obstipation, with vomiting. The symptoms which localize the lesion are tenderness in the right lower quadrant and spasm of the abdominal muscles. With these symptoms of local inflammation come symptoms of general bacterial infection, the most marked of which are increased temperature and increased white count, with perhaps chills. Cases of this sort are recognized by practically everybody, and practically every medical man advises immediate operation. If all cases were as simple as this there would be very few deaths from acute appendicitis.

But, unfortunately, a very large proportion of these cases of acute appendicitis are not so clear. In many instances the early pain is slight and temporary; often there is no vomiting and there may be practically no constipation. Local right-sided tenderness may be slight; spasm of the muscles may be so slight as to be recognized only by very experienced hands. As a result many cases of early appendicitis are "watched" for several days before a definite diagnosis is made. The inflammatory process in the appendix continues; much time is lost before operation is advised, and every surgeon is familiar with the fact that pa-

tients often are brought to operation after suppuration is advanced when the difficulties and dangers of the operation are unnecessarily and enormously increased.

Practically all cases of acute appendicitis are first seen by the medical man, and delay in presenting these cases for early and immediate operation rests very largely at his door. It may happen that the practitioner delays in recommending operation or in referring the case to the surgeon, thereby acquiring the reputation of being "conservative," to the great detriment of the patient. It may be said, in the first place, that appendicitis is *always a surgical disease and never a medical one.* It is perfectly true that a person may have an attack of appendicitis and have no subsequent attack; it is true that one attack of appendicitis may not lead to perforation and abscess formation. In other words, a given attack may "quiet down." But that is not the usual story. Nearly all cases, even in mild appendicitis, result in one of two things: in the vast majority of cases there are repeated mild attacks ultimately resulting in an attack so violent that the most conservative recognizes that operation is a necessity; or the patient is left with a chronic appendix surrounded by adhesions with resulting long-continued symptoms of chronic indigestion which may continue in some instances many years. I emphasize this end-result of conservatism in order to emphasize the necessity of early diagnosis and immediate operation. Don't wait in cases of appendicitis. It used to be recommended to wait in cases of appendicitis in which a so-called "appendix cake" had formed and in which operation had not seemed urgent, until the attack had quieted down, in the hopes of doing the operation in what was called the "interval." That always is erroneous and bad judgment. That is like hoping that the drop in a case of pneumonia will come on the third day; it may come, but is more likely not to. In the same way delay in operating on an acute appendix may lead to the formation of a large abscess which may suddenly rupture and cause a diffuse fatal peritonitis. There are two instances in which the surgeon does not have to exercise judgment, one is in a case of recognized appendicitis, that means immediate

operation; and the other is in the case of tumor in the breast; this always means an operation sufficient to make a microscopic diagnosis. No one can tell from the clinical symptoms exactly what the pathologic condition in the belly is. I have for a long while made it a point to attempt to foretell what I shall find on operating on a case of acute appendicitis; 90 per cent. of the time one can make a reasonably accurate forecast, but the other 10 per cent. of the time the symptoms correspond so little with the pathologic conditions as to make it a joke. Therefore, do not wait in cases of appendicitis.

As to the operation itself: the operation done within twenty-four hours is usually simple, uncomplicated, almost trivial. An operation done on the third or fourth day after the case has been "studied" by the general practitioner is often difficult, complicated with acute adhesions or with suppuration, and the risk of the operation is enormously increased; even then in practically all cases operation is safer than expectancy. Delay is almost as foolish as delay in a case of acute osteomyelitis with the hope that nature will establish drainage. Nature may, but in the vast majority of cases the patient dies of acute sepsis while waiting upon nature.

One thing is always to be borne in mind in cases of acute abdominal pain: the *probability* of the case being one of appendicitis. In other words, it is up to the first man who sees the case to prove it is *not* appendicitis rather than to demonstrate beyond doubt that it *is* a case of appendicitis. In cases of acute abdominal pain, remember that the percentage always is in favor of appendicitis. Continued severe abdominal pain always points to an organic lesion, especially if there are any symptoms of intestinal stasis. It is also to be remembered that appendicitis outnumbers any other acute abdominal lesions ten to one. Therefore, if in all obscure cases a man makes and sticks to the diagnosis of appendicitis he will come out with a very large percentage of correct diagnoses, although he perhaps will not make an intelligent, scientific diagnosis, but his average will be high. At any rate, the chief thing to bear in mind is the probability of an acute abdominal attack being due

to acute abdominal infection with the percentage enormously in favor of appendicitis, and in cases of recognized acute abdominal infection it is up to the doctor to prove that it is not appendicitis rather than to delay for the sake of making an absolutely certain diagnosis.

What are the most important symptoms? Localized tenderness and abdominal spasm. The tenderness in many marked cases is very slight. The spasm is the symptom most consistently present. Recognition of slight spasm is not always easy. It is much more likely to be recognized by the surgeon who is everlastingly testing for spasm than it is by the general practitioner. Many practitioners in my experience have been unwilling to accept as spasm anything less than absolute rigidity of the abdominal wall. That is not essential. Therefore, in cases of doubt where there is abdominal pain the case should be examined by a surgeon as soon as possible. He is much more likely than the medical man to recognize the slight differences in muscular rigidity. Moreover, it is *possible* to have an acute appendicitis with perforation and a diffuse peritonitis with neither pain, tenderness, spasm, constipation, vomiting or high white count. Fortunately, such cases are not common. They usually represent an acute gangrenous appendix in which perforation has taken place high up in the appendix close to the cecum with an overwhelming infection of the peritoneal cavity by the intestinal contents. Those cases, fortunately, are rare and usually give a history of a preceding acute attack, the pain of which quiets rapidly, leaving the patient extremely ill. These cases usually can be recognized by the history of the preceding acute attack with a subsequent severe illness associated with a "peritoneal look," difficult to describe, but easily recognized when once it has been seen.

As to the operation itself: I believe that the "right rectus incision," so called, is the best. I know that many men prefer McBurney's incision, but the right rectus incision has many advantages. It can easily be extended. If the case is without suppuration it gives perfectly good protection against hernia. If the drainage is necessary it can be perfectly well established.

The chance of hernia is no greater than after McBurney's incision in drained cases.

The recognition of the classic case is simple. No one should be excused for missing it. The diagnosis in about one-third of the cases may not be certain and often is difficult. The difficulties are greater, I think, in children than in adults. This appears to be due to the fact that for some unknown reason in children protective adhesions do not form around the appendix so readily as in adults. Also perforation seems more common in children than in adults, but until perforation occurs the symptoms may be comparatively mild. But after perforation the patient, who up to that time has seemed to be not seriously sick, may be rapidly overwhelmed by a diffuse peritonitis.

The 3 cases shown this morning illustrate some of the difficulties in making a diagnosis of appendicitis, particularly in children. Most cases of appendicitis are obvious; the obscure cases may call for the exercise of very great surgical judgment.

Case I shown is presented *after* operation. The child, M. C., a girl ten years of age, was brought in late last night. The family history is of no consequence. Patient has had measles and frequent attacks of tonsillitis, otherwise up to the beginning of this attack the child has been well. About 6 P. M. last night she suddenly began to have severe pain at the pit of her stomach and vomited several times, and at the end of three hours was quite light-headed and delirious. She arrived at the hospital at 11 P. M., no definite diagnosis having been made. Patient was restless and irrational, with a temperature of 105° F. The lungs were resonant throughout. There was no dulness; breath and voice sounds were normal. The heart was not enlarged and there were no murmurs. Her chief complaint was a pain in the abdomen in the right lower quadrant, which was very tender, with much rigidity and spasm. From the vomiting and abdominal symptoms it appeared like an obvious appendicitis, but the sudden onset and the high temperature, and the fact, as the surgeon who saw the child said, she did not "look peritoneal," led to a consultation with one of the visiting medical men, who, after careful auscultation and percussion, could find

nothing in the lungs. There was no sputum and no cough. Under those circumstances immediate operation was decided upon, and an appendix slightly hyperemic was removed through the right rectus incision. Temperature dropped to 101° F. this morning and the pulse dropped from 130 to 120. Urine examination showed urine to be amber, acid, 1020, slightest possible trace of albumin, with very few granular casts and an occasional white blood-corpuscle.

As you see, the child at the present minute is sick. Much too sick for a child after an operation for simple acute appendicitis without suppuration. Examination of the lungs this morning shows nothing abnormal. It will be surprising, I think, if this child later does not show either some complication or some other disease than appendicitis.

(This child continued very sick. Histologic examination of the appendix showed "healed appendicitis," which is a term we are accustomed to apply to appendices which show evidence of earlier inflammation. The *x*-ray on the second day after operation showed "bronchopneumonia." The temperature rose to 103° F., never went below 100° F.; delirium continued and the child died on the sixth day after operation. There was no autopsy.)

It should be borne in mind that the early stages of pneumonia may very closely resemble the beginning attack of appendicitis. For that reason a careful examination of the chest should be made in every case of suspected appendicitis, especially in children. But even that examination in cases of a central pneumonia may fail to show consolidation or râles. I have in one instance operated on a woman of thirty whose chest showed nothing before operation, but who showed on the second day after the operation a typical frank lobar pneumonia which ran a typical course, had lysis on the sixth day after operation, and recovered. I am not proud of the operation, but the experience has made me extremely careful to examine the lungs of all cases of suspected appendicitis. There is no record that any white count was done before the operation, and it seems to me this is one of the few cases where a white count might have settled the

probable diagnosis. White count in appendicitis seldom is above 15,000. In the beginning of pneumonia the white count is frequently above 20,000.

Case II.—P. B., a boy seven years old, who comes in with a complaint of pain on the right side of the abdomen. The family history appears of little consequence. The child has had measles and whooping-cough, but was well up to three days ago. Two days ago the child woke complaining of and crying with pain on the right side of the abdomen below the umbilicus. There had been no vomiting and the bowels have moved spontaneously. There is no history of any preceding attack. The child was at once sent to the hospital, and on arrival complained of slight general pain in the lower abdomen. There was general spasm of the abdomen, rather greater on the right-hand side, and there was slight tenderness on deep palpation, slightly more on the right side also. Temperature was 99° F., pulse 85, white blood count 10,000. The child was at once given an enema; the bowels moved and *all* pain and tenderness disappeared. The patient was comfortable and slept all night. Yesterday morning there was a little spasm on the right lower quadrant, but no tenderness or pain. This morning a slight amount of spasm persists; there is no tenderness or pain, even on deep pressure; temperature is 96.6° F. Because of the persistence of the muscular spasm and because the patient is a child it seems unsafe to wait any longer. Appendicitis in children is often quite different from appendicitis in adults. For that reason it is particularly desirable that the diagnosis in children be made early, and with children, in case of doubt, it is much safer to operate than it is to delay. Protective adhesions seem to form around the appendix in children much less commonly than in adults, and if the inflammatory process continues and leads to perforation, an extensive general peritonitis may arise in a very short time, although up to the time of perforation the symptoms may be slight. In this instance the operation is done for these reasons: because of an attack of severe abdominal pain, right sided, and because of the persistence of spasm. In an adult I should wait for

examination by *x*-ray. In this particular case it seems imprudent.

(Operation was at once done through a right rectus incision. The appendix was found 8 cm. long, very much injected and distended. There was no perforation and almost no adhesions. There was a small amount of free, clear fluid. The wound was closed tight. The child made an uninterrupted recovery. The pathologic report was "early healing appendicitis.")

In this case it is possible that the inflammation would have subsided without operation. It is just as certain that other attacks would have occurred, and it also is possible that the inflammation, if continued, ultimately might have led to a sudden acute perforation and a sudden diffuse peritonitis.

Case III.—B. S., a Jewish girl, thirteen years old. Patient came to the hospital ten days ago complaining of attacks of pain in her right lower quadrant, and stated that she had had similar attacks at irregular intervals during the past two years. During these attacks the pain was quite severe; attacks never lasted over a few minutes. There has been no vomiting and the child was moderately constipated. The lungs and heart were negative. Menstruation had not begun. Temperature 98.6° F.; pulse 90; white blood count 8200. Urine negative, containing no albumin, blood, or casts. On account of the child's age no vaginal was done. Examination of abdomen was absolutely negative. Neither pain, tenderness, nor spasm was evident and the two sides were alike. *x*-Ray examination showed a moderate amount of stasis around the cecum; appendix was visible; there was no evidence of gall-stones or of stone in kidney or ureter. The *x*-ray people made a diagnosis of "chronic appendicitis," based upon the stasis and the visibility of the appendix.

The child was under observation for ten days before being operated upon. The spasmodic pain at irregular intervals might have been from gall-bladder, stomach, kidney, or ureter. The only positive evidence in favor of appendicitis was the *x*-ray.

(The pathologic report on this case was "healed appendicitis." The wound was closed, sewed up tight, and patient made an un-

eventful recovery. Up to date there has been no recurrence of symptoms.)

This case illustrates one clinical fact very well, and that is this, as a matter of percentage appendicitis outnumbers other abdominal lesions at least 10 to 1. Practically all cases of appendicitis are at some time or other accompanied by abdominal pain. In cases of indefinite abdominal pain in which no certain diagnosis can be made the percentage always is in favor of appendicitis. Many cases of appendicitis never give the classic symptom-complex of lower right quadrant pain, tenderness and spasm, with vomiting, constipation, some rise of temperature, and usually increased white count. Therefore, in the vague cases, always bear in mind the probability of appendicitis; and there are many cases of abdominal pain where it is perfectly desirable to operate on the history alone.

CLINIC OF DR. WILLIAM H. ROBEY, JR.

BOSTON CITY HOSPITAL

ANEURYSM OF THE DESCENDING AORTA

Etiology; Diagnosis; *x*-Ray Findings; Complete Results of Autopsy and Discussion of Treatment.

EVEN a casual glance at the literature is sufficient to call the attention of the physician to the frequency with which syphilitic infection involves the arterial system, yet it is not uncommon for the internist, who sees more of the so-called tertiary lesions than any others, to find patients who have had no antisyphilitic treatment for many years, and have sought the internist's aid because of symptoms of circulatory disturbance. Within a week an intelligent man consulted me because of a dilated arthythmic heart and arteriosclerosis who had undergone a course of antisyphilitic treatment many years before under the direction of a distinguished syphilographer. When I questioned him about subsequent treatments he replied that nothing had been said to him about that, and since he had been perfectly well for many years he had not felt the need of medical advice. I have had this experience many times. Anders (Amer. Jour. Med. Sci., Dec., 1915, vol. cl) says that the intimate connection between lues and aneurysm was well known to the older writers (Pari, Larcisi, and Morgagni), but it has been especially emphasized by many modern writers. So, while the facts have been known for many years, their importance has not taken hold of the minds of physicians in general. The diagnosis of syphilis should be made at the earliest possible moment by use of the dark-field illumination and the Wassermann test. Fordyce (Amer. Jour. Med. Sci., Oct., 1916) says, "It cannot be emphasized too frequently nor too

emphatically that the fate of the syphilitic individual depends largely upon the early diagnosis of his infection and the intensity with which his treatment is carried out in the first six months. It is in the accomplishment of this purpose that the modern aids to diagnosis have rendered such invaluable service." Fordyce adds that even with this knowledge mistakes are made and patients given an assurance wholly unfounded, the error not being discovered until it is too late. Knowing then that the Spirocheta pallida is the cause of a large number of cases of aortitis, it is our duty to search the secretion of all venereal sores. With reasonable training the dark-field illumination is easy of application.

In more recent years the Wassermann reaction has become a routine. In this hospital and many others it is taken in every case. At first the blood Wassermann was made, but now in a considerable number of cases there is also a test of the spinal fluid. In private practice we have been too apt to have a Wassermann done only when we suspected a previous syphilitic infection. Our hospital experience with the Wassermann test should make us wiser in the management of our private patients. Lewis states that syphilis was recognized by the older of the present-day physicians as being a most frequent and important deleterious factor in the health of the community, and quotes Osler's statement with reference to the general diagnosis of the disease: "It is to be remembered that syphilis is common in the community, and there are probably more families with a luetic than with a tubercular taint."

Etiology.—Most writers are of the opinion that syphilis is the common cause of aneurysm. Anders lays stress on the position of the aneurysm in distinguishing its etiologic variety, claiming that the ascending portion of the aorta is the usual seat of luetic aneurysms. The greatest number occur in the ascending portion and the smallest in the descending.

Various observers have reported 60 per cent., 80, 82, 85, and 65 per cent. due to lues. Anders in 621 cases found 58.5 per cent. of luetic origin, but considered the percentage too low, since some of the cases had not had the Wassermann test.

Allbutt considers syphilis to be the common cause of aneurysm, and Osler places the percentage as high as 80 or 85, and stated that nowadays it is rare not to find a positive Wassermann reaction in an aneurysmal patient under fifty. Osler believed that the specific fevers cause areas of degeneration in the aorta not uncommonly, but fortunately, in most instances, they are confined to the intima, but occasionally, as in typhoid fever, may cause changes in the media. He believed that infections other than syphilis play a minor rôle in the causation of aneurysm. Mallory says that tuberculous lesions of the arteries and aorta have in rare instances caused weakening of the wall and aneurysmal formation. Tuberculous lesions are very common in the capillaries and lead to complete occlusion of them. They occur more or less frequently in the small veins and arteries, especially in certain parts of the body, such as the lungs, for instance, and are dangerous because the tubercle bacilli may multiply in great numbers, and by escaping into the circulation give rise to acute generalized miliary tuberculosis. Rarely they lead to the formation of an aneurysm in arteries or the aorta, from which again large numbers of bacilli may be discharged into the blood (Principles of Pathologic Histology, 1914). Another point which I wish to call to your attention is emphasized by Harlow Brooks in an article on the heart in syphilis. He believes that it is unsafe to attempt to divide syphilis into periods or stages, since one commonly finds so-called tertiary lesions appearing in secondary stages, and from time to time in the tertiary period sudden exacerbations of the process spring up which can only be compared to those which we are accustomed to ascribe to the secondary stage. Brooks also calls attention to the early appearance of syphilis of the aorta in patients who have been thoroughly treated at the time of their initial lesion. It has been my experience with the majority of cases of syphilis of the aorta that the condition appears ten to fifteen years after the original infection. I remember a young man whom I saw with a colleague and who was treated thoroughly at the time of his initial lesion. He was lost to us, but fourteen years later was admitted to the hospital with well-marked aortic regurgitation.

These points of etiology and treatment I have dwelt upon particularly because they are well illustrated in the 2 cases I have to show you.

Diagnosis.—The early diagnosis of aneurysm is always difficult. Aneurysm of the descending thoracic aorta is often overlooked. Osler says that it is frequently latent and that pulmonary and pleural symptoms are common. Pain in the back is the commonest symptom and is generally severe; dysphagia is not infrequent. They may reach an enormous size, as is well illustrated by our patient.

The first case is a white man sixty-three years old. He came into the hospital in 1908 with a fractured femur. At that time he had tortuous and thickened vessels and a systolic murmur at the aortic area. He was admitted to the medical side of the hospital on June 12, 1919 from the Boston Consumptives Hospital where he had been for one year and four months. His family history was unimportant. His wife died at thirty-seven in childbirth and his only child died at the same time. The chief complaint was pain in the right hip and left chest existing for two years. He was unable to recall any diseases of childhood or adult life and was never sick in bed until he went to the Consumptives' Hospital. There was no history of precordial pain, vertigo, hemoptysis, chills, night-sweats, or edema. At times he felt feverish and for a year had malaise and pain in the left side. The night urine exceeded the day. The appetite had been fair and until one year ago the bowels regular. He denied having any venereal infection. His habits were good. He smoked moderately and did not use alcohol. He had always done heavy work. Best weight 225 pounds eight to ten years ago, 175 pounds one year ago, and 170 pounds now. Two years before admission, while lifting a heavy casting in the Navy Yard, he felt a sudden strain in the right lumbar region. He was strapped by the yard doctor and continued work. The pain was persistent, but never interfered with sleep. At the time of admission to the Consumptives' Hospital there was slight cough, with morning sputum, and the patient was placed in a cottage ward for the slightly incapacitated. There were no signs of active tuberculosis

for one year before admission to the Boston City Hospital, but three months before he had severe pain in the left hypochondrium. The pain was without relation to meals, did not radiate, and was not increased by breathing. He complained of general weakness, pain in the left hypochondrium (three months' duration), poor appetite, and constipation.

The general examination showed a well-developed, somewhat emaciated man lying comfortably in bed, without apparent distress. He answered questions readily and intelligently.

The head examination was essentially negative, while the expansion of the chest was good.

The lungs were dull throughout except in the left base, where the note was flat. Tactile fremitus and voice sounds diminished, but absent at the left base. Over the entire left chest, front and back, there were medium and coarse crepitant râles. In the right apex there were a few crepitant râles after coughing.

The heart was enlarged, 3.5 cm. to the right of the midsternal line and 13 cm. to the left. The cardiac impulse was seen and heard best in the fourth space just outside the nipple line. The sounds were strong, with an occasional dropped beat. At the aortic area the second sound was accentuated. No murmurs. The vessel walls were slightly thickened and beaded. The brachials were tortuous. The abdomen was slightly rounded and tympanitic; no masses or tenderness except in the epigastrium. The liver and spleen were not palpable. The neurologic examination was negative. The extremities merely showed the results of old injuries.

While the patient was at the Consumptives' Hospital an *x*-ray plate of the chest was taken which showed a large shadow apparently connected with the heart.

The *x*-ray plate taken June 20, 1919 at the City Hospital (oblique view) showed a greatly enlarged transverse and descending aorta. The posterior mediastinum appeared to be clear. There was a large shadow in the left lower chest which was difficult of interpretation. The *x*-Ray Department suggested aneurysm of the thoracic aorta, and growth, either from liver or from some organ in the chest. The physical signs remained prac-

tically unchanged. The percussion note was almost flat over the left lower lobe behind. The left axilla, however, had normal resonance, so that fluid may be ruled out. There were many fine râles over this area and the breath sounds were markedly diminished. Fremitus was diminished and there was no Grocco sign. The patient had lost a great deal of weight, as was manifested by the flabbiness of his legs. There was a more noticeable and distinct shaking or shock of the patient's whole body with each heart-beat. This shock seemed to be entirely out of proportion to the heart impulse, which was barely felt in the fourth space just outside the nipple line. Just above the nipple in the second and third spaces and separate from the apex impulse was a diffuse pulsation synchronous with the heart rate. The percussion note seemed slightly dull over this, but was far less dull than the note a few centimeters nearer the nipple line. The liver edge could be distinctly felt 4 cm. below the costal margin in the nipple line. No pulsation could be felt in the abdomen even when the fingers were pressed deeply into the epigastrium. There was no evidence of an aneurysm in either groins or popliteal spaces. Patient had begun to be slightly dyspneic. He complained of pain only in his right hypochondrium, and he explained this due to a kick in the ribs which he received from a horse two years ago.

Bismuth series were done on the patient to show the relation of the esophagus to the tumor. Anterior plates showed the esophagus crossing the chest from right to left and entering the cardia of the stomach in the extreme left side of the body. An oblique plate showed esophagus pushed anteriorly by a big rounded mass. The interpretation of the *x*-ray and clinical findings were difficult. The mass may be either below or above the diaphragm. There is no question that the transverse and descending aorta were markedly enlarged. With this positive factor it seemed likely that the diagnosis of aneurysm of the thoracic aorta was the correct one. The patient's blood and urine were essentially negative. Wassermann negative. Patient failed rapidly and was markedly cyanosed. Had not developed hoarseness.

Discharged, dead, June 23, 1919.

Diagnosis: Aneurysm of the thoracic aorta.

Autopsy, June 23, 1919. Fourteen hours postmortem. Body length 175 cm. Age sixty-three years. Body—L. E. Sutton. Head—not done.

Clinical Diagnosis.—*Aneurysm.*—The body is that of a well-developed and fairly well-nourished white man. Rigor mortis present, lividity of dependent portions; pupils equally dilated 5 mm. in diameter. No edema or glands palpable.

Peritoneal Cavity.—Diaphragm extends on the right to the fourth interspace; on the left to the sixth rib. The diaphragm on the left is pushed down into the abdominal cavity by a large tumor mass which extends from the thoracic cavity to about opposite the umbilicus, separating the crura. The liver is displaced to the right and extends down even with the umbilicus in the right midclavicular line. The left border of the liver extends to the left costal cartilages 3 cm. to the left of the ensiform cartilage. The stomach and pancreas lie anterior and on top of the tumor mass; the spleen to the left and a few centimeters lower than normal. The left kidney is found to be posterior to this mass, while the right is covered by the liver. The lower poles of both kidneys extend to the line joining the posterior superior spine to the ileum. There is a slight amount of bloody fluid in the cavity (about 60 to 100 c.c.). The serous coat of the intestines is somewhat blood stained. The appendix is retrocecal, free of adhesions, and essentially normal. The mesenteric lymph-nodes are not enlarged.

Pleural Cavities.—The left cavity contains many fibrous adhesions joining the lung to the posterior and lateral walls, also the base of the lung to the projecting tumor mass which is posterior to the pericardial sac and occupies about at least one-third the cavity. On the anterior surface of the fifth and sixth ribs opposite the angle are found small hard, calcareous nodules about ¼ to ½ cm. in diameter, projecting into the pleural cavity. The right cavity is free of adhesions. The pleura is smooth and glistening. The seventh and eighth ribs show calcareous nodular elevations.

Pericardial Cavity.—It contains about 50 c.c. of straw-colored fluid slightly blood-tinged; otherwise negative.

Heart.—Weight 415 grams. The epicardium contains fat. The muscle wall is firm and red, with brownish tinge. The mitral valve shows thickening. The other valves are negative. The foramen ovale is not patent. The coronaries are patent, the intima containing a few yellowish patches.

Lungs.—The right lung is crepitant throughout, except the posterior portion, which is dark red in color, and on section blood escapes from the cut surface. The rest of the lung is grayish in color and practically bloodless. The left lung is similar to the right, although about one-half the size. The bronchi and blood-vessels are negative. The bronchial lymph-nodes are anthracotic.

Spleen.—Weight 145 grams. There is a small accessory spleen about 1 cm. in diameter. The capsule is slightly wrinkled and grayish in color. Beneath the capsule and throughout the tissue are small pin-point grayish areas. On section, it cuts with resistance, revealing a firm pulp with a reddish mottling. The trabeculæ are thickened.

Gastro-intestinal Tract.—The stomach is contracted and contains very little fluid. The mucosa of the transverse band shows congestion; otherwise negative. The duodenum shows much congestion; the serous coat is dark red in color as well as the mucosa. The jejunum and ilium essentially negative. Large intestine and rectum negative.

Pancreas.—Slightly smaller than normal. The superior border shows congestion. It is attached to the tumor mass by blood-stained fibrous adhesions; otherwise negative.

Liver.—Weight 1855 grams. The surface of the liver is smooth and glistening, save for lower portion of the right lobe, which is finely granular in character. On section the tissue is firm and has the characteristic reddish-brown and grayish mottling, giving the appearance of the nutmeg liver. The gall-bladder is distended and filled with yellowish viscid bile. There are three small calculi, faceted in shape, measuring about 1 cm. in diameter. The length of the bladder was 10 cm. and the circumference about 5 cm. The common bile-duct is patent.

Kidneys.—Weight 175 grams apiece. The capsule strips readily. On section, the tissue is firm. The cortex measures 6 mm. The cut surface has a grayish appearance. The pyramids are injected.

Adrenals.—The adrenals measure about 5 x 5 x 1 cm. respectively. They are negative.

Bladder.—The walls are not thickened and mucosa not injected. Negative in appearance.

Prostate.—Normal in size and negative.

Testicles.—The tubules do not string out; negative.

Penis.—Negative.

Aorta.—Greatly dilated in the decending portion, forming a large tumor mass, which measures 25 x 23 x 13 cm. This tumor mass, including the aorta, as well as the esophagus and stomach, weighs 3500 grams. Its outer surface is tough, firm, and blood-stained and has attached to it by fibrous adhesions the esophagus, stomach, diaphragm, and portion of lung. The esophagus enters the tumor at the superior border and the cardiac end of the stomach is found at the inferior border. On opening the esophagus it is traced along the anterior surface of the tumor mass, making a half-spinal curve. The mucosa is grayish in color, showing no signs of hemorrhage. It measures 5 cm. across. Following the lumen of the aorta it is found to pass through the tumor mass to the left of the center, having a wall 13 cm. thick on the right and 8 cm. thick on the left, which is quite firm, dark gray in color, and showing well-marked laminations. There are areas which are reddish in color and more friable. The lumen through the mass varies from 4 to 5 cm. in diameter. The intima is smooth and dark red in color. Running vertically within the intima are threads of grayish tissue. Attached to the intima in places are soft dark red, jelly-like postmortem clots. The ascending aorta is dilated. The intima contains yellowish raised areas which more or less pinch it, producing a wrinkled appearance. This thickening of the intima is found throughout the aorta. The descending aorta from the arch to the tumor mass is gradually dilated, giving it the shape and volume of about a 500-c.c. Erlenmeyer flask. The tumor mass extends to 8 cm.

of the iliac arteries. The circumference of the aorta here is 6 cm. The intima shows yellowish plaques. The arteries leading off from the aorta are dilated.

Anatomic Diagnosis.—Aneurysm of descending aorta (thoracic and abdominal). Anrtitis and arteriosclerosis. Chronic passive congestion of liver and spleen.

Histologic Examination.—The heart is negative. The valves are thickened, due to sclerosis. Aorta: The intima shows marked sclerosis and some hyalinization. The thrombosed wall of the dilated aorta consists of layers of fibrous deposits containing coagulated serum within its meshes. No Treponema pallida were demonstrated in the heart muscle, liver, aorta, or portions of the thrombosed wall of the aneurysm. The postmortem Wassermann was negative.

This patient's history and examination showed many interesting features. It is remarkable that his descending thoracic aorta should have passed through an organized aneurysm, larger than a man's head, causing the esophagus to pass well to the right in a half-spiral curve and yet giving almost no symptoms. No wonder that the left lung was compressed to half its size and that the physical signs led to a diagnosis of pulmonary tuberculosis which, however, was never substantiated by finding the tubercle bacillus nor by autopsy. Osler calls our attention to the indurative changes in the lung which may follow compression by an aneurysm. Is it not surprising that with the esophagus so much out of place there was never painful deglutition? This patient worked up to sixteen months before his death and was never in great discomfort at any time. He was sent to the Boston City Hospital from the Consumptives' Hospital for diagnosis. The small aneurysm in the transverse aorta was found on physical examination and first called our attention to the possibilities of the larger mass. It shows how large an aneurysm of the descending aorta may exist without symptoms.

What was the cause of this aneurysm? Syphilis is the accepted etiologic factor, as already stated, yet by all tests the patient had never been infected with the Treponema pallida. Other infections rarely cause aneurysm and tuberculosis oc-

casionally, but the examinations gave no evidence of tuberculosis. Osler gives "strain" as the second determining factor, particularly the internal strain associated with sudden and violent muscular effort. "The media is the protecting coat of the artery, and during a violent effort, as in lifting or jumping, laceration or splitting of the intima may occur over a weak spot. If small, this leads to a local bulging of the media and the gradual production of a sac, or the tear of the intima may heal completely, or a dissecting aneurysm may form. In other instances a wide-spread mesaortitis leads to a gradual, diffuse distention of the artery. This type of aneurysm, frequently seen in the aged, may follow ordinary chronic atheroma." Our patient was subjected for many years to violent muscular strains. It is not likely that emboli could have caused the aneurysm, since his valves at autopsy were practically negative. Death is not commonly due to rupture of the aneurysm even in the sacculated form, and, of course, in an organized aneurysm such as our patient had rupture is impossible. During the few days he was in the hospital he gradually failed, as is the rule with a degenerated heart and blood-vessels. He became more and more cyanotic, with failing pulse and increasing stupor. When asked how he felt he always replied that he was doing well and was without discomfort of any kind.

The treatment of these cases of advanced aneurysm is the same as for degenerated heart and arteries. Absolute rest and freedom from exertion are imperative.

The second case illustrates the beginning of an aneurysm of the descending aorta. The patient, aged forty-five, is a restaurant keeper. He is a large, well-nourished and well-developed man and has never been ill.

When he says he was never ill he means ill in bed. He has had, however, two serious infections—syphilis and gonorrhea. At the time of his syphilitic infection seventeen years ago the diagnosis was prompt, and the treatment thorough, lasting three years. Seven or eight years ago he had one injection of salvarsan. In the summer of 1918 he received three injections. Eight to ten years ago he began to notice a pain in the back and shortness of breath when going up an incline. He is very fond of dancing

but after going around the hall twice he becomes so short of breath that he has to stop. Four years ago he had an *x*-ray plate of his heart which disclosed no abnormality, but another plate within a few weeks shows a slight enlargement and bulging of the descending aorta. I have examined him twice, but even after seeing his second plate I was unable to make a diagnosis of aneurysm of the descending aorta from the physical signs. There is no evidence of other aneurysms. His color is good, the heart area is normal; at times there is a soft systolic murmur at the apex not transmitted. There is no throbbing of the vessels and no accentuation of the aortic and pulmonic second sounds. The blood-pressure is 120 systolic and 90 diastolic. The pulse is regular, 60 in rate, and 72 after exercise. A thorough examination on two occasions revealed nothing else.

This man had a clear history of syphilis seventeen years ago, with thorough treatment then and at intervals since. He has no evidence of a general atheroma. He began first to notice symptoms of circulatory involvement seven years after his initial infection, and this in spite of excellent treatment. His symptoms have been slight, but they are greater than in the previous patient, whose tremendous aneurysm gave little indication of its presence until a year and a half before death. They perhaps represent the extremes of aneurysm.

Treatment.—In the first case there was little to offer. At the time we saw him he was *in extremis* and died within a few days from circulatory failure. Had he been seen years before and the diagnosis established, care of the heart and blood-vessels with the prevention of subsequent infections would have been indicated. The second patient has already improved. He had been improperly masticating his food and has received instructions which have been of assistance to him. By reducing his activities along certain lines strain has been removed from his heart and great vessels. He reports that he is practically free from pain now. Careful antisyphilitic treatment must be given at regular intervals.

CLINIC OF DR. EDWIN A. LOCKE

PNEUMONIA SERVICE, BOSTON CITY HOSPITAL

EMPYEMA COMPLICATING PNEUMONIA

Case I.—Lobar Pneumonia (Type I); Antipneumococcus Serum; Empyema (Type I Pneumococcus and Bacillus Influenzæ); Rib Resection and Recovery.

Case II.—Bronchopneumonia (Type IV, Pneumococcus Streptococcus Hemolyticus, Bacillus Influenzæ); Empyema (Streptococcus Hemolyticus); Rib Resection and Recovery.

I SHALL discuss 2 cases of infection of the pleura complicating pneumonia which illustrate the two common types of empyema, namely, the pneumococcus and the streptococcus forms.

CASE I

O. S., a man aged thirty-one, entered the service February 28, 1920. Occupation, locomotive engineer.

Family and **past histories** good.

History of Present Illness.—Seven days before entrance the patient had a very severe chill, followed by headache, general malaise, high fever, and profuse sweating. At onset there was no cough or pain. Breathing became increasingly difficult and the prostration more pronounced. A painful, persistent cough developed on the second day, which has persisted, and for the past four days he has had profuse, greenish-yellow, mucopurulent expectoration. The cough and dyspnea were very marked at entrance.

Physical Examination.—A well-developed, robust man who seems desperately sick. The breathing is especially distressing, being both difficult and painful; orthopnea.

The face is injected and bathed in perspiration. Tongue very dry, coated, teeth with sordes, and throat injected and dry.

Heart borders 2 cm. to right and 8 cm to left of midsternum; action regular; sounds of good quality; no murmurs.

Chest symmetric, well formed, expansion equal. Dulness, intense bronchial breathing, bronchophony, increased tactile fremitus, and a few medium crackling râles over the right upper lobe. Otherwise the lungs are negative.

Abdomen negative. Extremities and reflexes normal.

Laboratory Reports.—Blood-culture and blood Wassermann reaction negative. Urine examination showed characteristics of a "fever urine" only. Blood-pressure 120/85. Phenol-sulphonephthalein excretion 70 per cent. in two hours. Sputum shows Type I pneumococcus. Bedside *x*-ray examination confirms the signs of consolidation in the right upper lobe.

Subsequent History.—Following the finding of Type I pneumonococcus in the sputum the patient's sensitiveness to horse-serum was immediately tested by 0.02 c.c. of horse-serum diluted 1 to 10 given intradermally. No local reaction occurred, but a desensitizing dose of 1 c.c. of the same serum was injected intramuscularly, and in two hours (5 P. M. February 29th) 100 c.c. of the antipneumococcus serum were given intravenously. No thermal or anaphylactic reaction followed.

During the twenty-four hours following the injection of immune serum the patient showed a very striking improvement and the temperature fell to normal. On the evening of the same day, however, the temperature again rose to 101° F., and subsequently ran an irregular course, as will be seen from the accompanying chart. The signs of consolidation in the right upper lobe rapidly disappeared.

In spite of improvement in the patient's general condition and the absence of any local symptoms, signs in the lower right back gradually developed. On March 6th the percussion note over the lower third of the chest posteriorly was very dull and the respiration of a moderately distant vesicular type. Fremitus was likewise diminished and a few medium râles were heard, but no friction sounds. *x*-Ray examination showed a dense shadow

suggesting fluid. An exploratory puncture was done in the seventh space, but no fluid was obtained. Culture negative. Improvement in the general condition continued, but the temperature followed a very irregular course and the signs in the

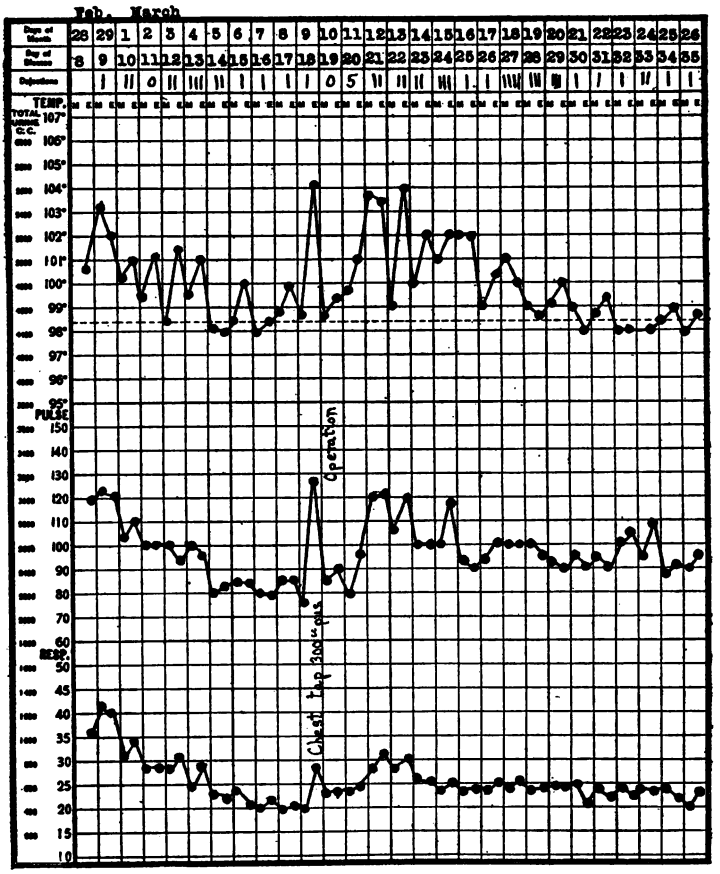

Fig. 52.—Case I.

right back became more suggestive of fluid. White count 12,200. On March 9th a second paracentesis was done in the seventh space just inside the posterior axillary line and 300 c.c. of thin pus withdrawn. Culture showed pneumococcus Type I and Bacillus influenzæ.

The case was transferred to the Second Surgical Service, and on March 10th, under local anesthesia, Dr. Hubbard resected a portion of the ninth rib, opening into the pleural cavity and obtaining a small amount of pus. Although drainage was satisfactory, no striking improvement followed the operation for some time. Roentgenograms made on the 15th showed clearly a fairly dense shadow in the middle of the right lung which strongly suggested encapsulated fluid. High in the axilla a considerable area of moderate dulness with tubular breathing and egophony likewise indicated the possibility of encapsulated fluid. Exploration of this area with a needle yielded only a single drop of pus, presumably from the lung, as the needle could be felt to enter solid tissue. Culture, Streptococcus hemolyticus. The condition was regarded as a probable complicating bronchopneumonia due to the streptococcus.

No further complications occurred, and convalescence progressed slowly until early in May, when the case was considered fit for discharge.

Aside from the complicating empyema this case is interesting from the point of view of the specific serum treatment given. It has become a routine procedure on the Pneumonia Service to treat all suitable Type I pneumonias with antipneumococcus serum, and the results during the past eighteen months seem to fully justify its use. Our series of treated cases is a small one, and without simultaneous untreated control cases it is impossible to form any accurate estimate of its value. A total of 75 cases have been treated by this method, with a mortality of 17.3 per cent. In 56 of those treated previous to the eighth day of the disease the mortality was only 12.5 per cent. The mortality in 57 untreated Type I cases was 26.3 per cent. The statistics of the Rockefeller Hospital on this point seem to leave little question of the merits of the serum treatment in pneumonia of this type. A mortality of 25 per cent. was shown in their series of Type I pneumonias treated without the immune serum, while in a group of 107 cases of the same type treated with serum the mortality was only 7.5 per cent. Similar figures of an equally convincing nature have been reported from other clinics. In the light of

these results one does not hesitate to take the stand that every case of Type I pneumonia should be given the advantage of specific serum treatment.

The procedure is not without danger of anaphylactic symptoms and certain precautions are necessary. An intradermal cutaneous test should always be made by injecting 0.01 to 0.02 c.c. of horse-serum diluted 1 to 10 and controlled by a similar injection of normal salt solution. As a further safeguard even when this test is negative an intramuscular injection of 1 c.c. of the serum to be used should be given to insure desensitization at least one hour before the intravenous injection. Should the cutaneous test be positive the patient must be desensitized before administering the serum.

The serum diluted with equal parts of normal salt solution and at body temperature is given intravenously in doses of 100 c.c. At least fifteen minutes should be allowed for the first 15 c.c. and at least forty-five minutes for the entire amount. Unless a definite fall in the temperature and improvement in the patient's general condition results the same amount of serum is repeated every eight hours. The maximum amount given to any of our cases was 900 c.c., and the average for the 75 patients was 197 c.c. It is of the utmost importance that the serum be used as early as possible in the disease. If the patient develops symptoms of anaphylactic shock during the administration of the serum an immediate injection of 0.5 to 1 c.c. of adrenalin chlorid solution (1 : 1000) and atropin (gr. $\frac{1}{100}$) should be given. The so-called "thermal reaction," which frequently occurs soon after the intravenous injection, and the "serum sickness," which appears from one to two weeks later, while very distressing to the patient, are never serious. Finally, my experience with this procedure leaves me with the strong conviction that it should be used only by those thoroughly experienced in serum administration.

CASE II

W. A., male, aged fourteen, entered service February 15, 1920.

Family History.—Good. No history of tuberculosis.

Previous History.—Generally good health. Measles and

whooping-cough in early childhood; good recovery without complications.

History of Present Illness.—February 7th he got his feet wet and was chilled while playing in the snow. In the evening felt very chilly and had intensely sharp pain in right lower thorax, which was exaggerated by cough and deep respiration. Persistent, harassing cough, at first unproductive, but soon with thick mucopurulent sputum, which was occasionally blood streaked. Severe diarrhea at onset. Otherwise no noteworthy symptoms.

Physical Examination.—Normal physical development for age. Face deeply flushed; distressed, anxious look. Respirations very rapid and shallow.

Marked dulness over right lower lobe with tubular respiration, increase in whispered and spoken voice and tactile fremitus, many medium crackling râles throughout. Percussion note over the right upper lobe tympanitic, but no abnormality in the respiration or fremitus.

Heart normal size and position, apex-beat neither seen nor felt, sounds faint, no murmurs, rate 120 per minute. Pulse somewhat bounding, but of rather poor volume and tension.

Examination otherwise negative.

Report of Laboratory Examinations.—White count 26,000 per c.mm. Hg. 75 per cent. Blood-pressure 98/70. Blood-culture negative. Examination of urine and stools negative. Wassermann blood test negative. The sputum showed the presence of pneumococcus Type IV and bacillus influenzæ.

February 18, 1920: The dulness over the right lower lobe more marked than at entrance and a slight distant quality to the respiration; tactile fremitus unchanged. The signs suggested the possibility of fluid and an exploratory paracentesis was done at a point just below and outside the angle of the scapula. The needle could be felt in solid lung and no fluid was obtained. No organisms were obtained from the lung puncture.

February 20th: Temperature 103° F. Pulse 120. Respiration 30. Patient seems distinctly more toxic; very marked dyspnea. Very little movement of right chest with respiration.

Flatness has replaced the former dulness over the right lower lobe and the area is considerably more extensive. Over an area of about 2 inches in diameter at the site of the previous puncture the respiration and fremitus have a distinct amphoric

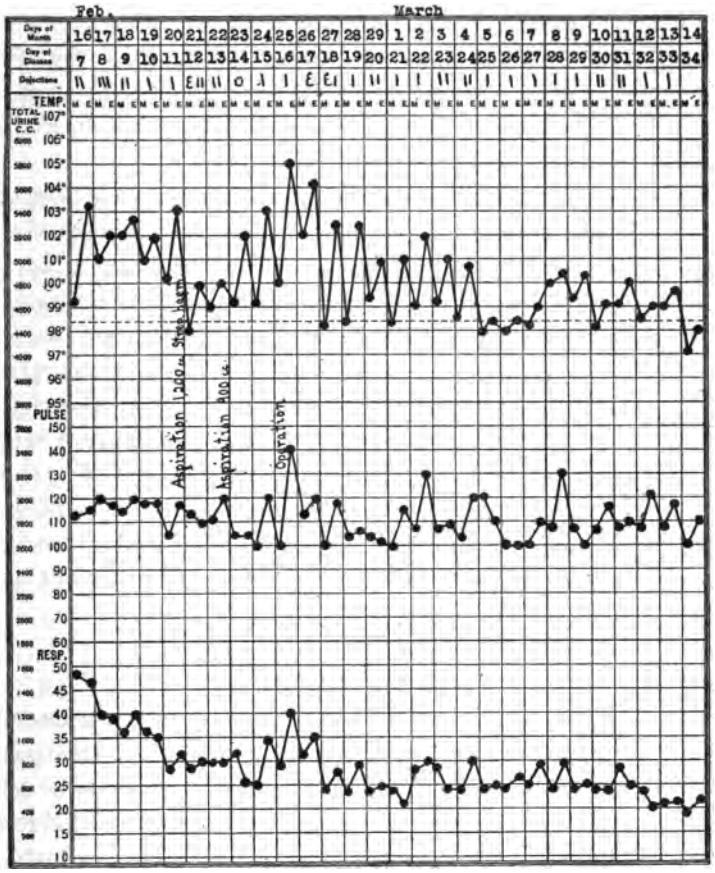

Fig. 53.—Case II.

quality, otherwise the respiration all over the lower two-thirds of the right chest is intensely bronchial in type, but with a somewhat distant quality. No râles. Percussion over the right upper lobe is frankly tympanitic and the respiration very loud, though vesicular in type. The heart does not seem displaced to left.

On this date, the eleventh day of the disease, a second aspiration was done and 1100 c.c. of thin pus was obtained. Cultures gave a pure culture of hemolytic streptococcus.

On the following day the temperature fell to 98° F., but rose to 100° F. in the afternoon. The patient showed a very striking improvement in his general condition however.

February 22d: The chest was tapped for the third time and 900 c.c. of thin pus withdrawn. Culture, hemolytic streptococcus. Following this procedure signs of extensive hydropneumothorax appeared.

February 25th: The patient was transferred to the Surgical Service of Dr. Hubbard and under gas-oxygen anesthesia a trocar was inserted in the eighth space posteriorly and a No. 28 French catheter introduced. Drainage was not satisfactory and subsequently a costectomy was done. The cavity then drained freely and the patient gradually improved, the temperature reaching normal on March 5th, as will be seen from the chart. For the week of March 7th to the 13th the temperature ran a somewhat irregular course, with a maximum of 100° F. Convalescence was slow, but with steady improvement. The pleural cavity drained freely, but with gradually diminishing amount until early May, when it entirely subsided. The patient was discharged well on May 15th.

TYPES OF EMPYEMA

A clear understanding of the types of pneumonia is necessary to a proper appreciation of the problems affecting the treatment of empyema. Pneumonia as usually seen assumes two very distinct forms: (1) the lobar pneumonia, which is usually due to the pneumococcus, and (2) the bronchopneumonia, which is almost invariably of streptococcus origin. Except in the case of children the type usually met with in civil life is a primary lobar pneumonia. The second type of the disease is seen chiefly in epidemic form such as occurred so commonly in the cantonments during the past few years and especially as a secondary infection in measles and influenza. This type very often follows an infection of the upper respiratory tract, has a relatively slow

onset, very severe symptoms, frequent complications, and high mortality.

The infecting streptococcus may be of the hemolytic or non-hemolytic variety, but the former is more common and the infection more severe.

The two case histories which I have given this morning illustrate very well these two varieties of pneumonia. The first was a typical lobar pneumonia of pneumococcus origin, the second a severe bronchopneumonia due to the hemolytic streptococcus. It is interesting that in the first case there was a complicating streptococcus bronchopneumonia as sometimes occurs.

Similarly, it is important to differentiate two general types of empyema. While infection of the pleura may be due to a considerable variety of organisms, the vast majority readily fall into two groups, namely: (1) pneumococcus empyema following lobar pneumonia, and (2) streptococcus empyema complicating bronchopneumonia. As in the case of the two types of pneumonia, the second is usually more severe and consequently more commonly fatal. Among our cases of empyema 50 per cent. were due to the pneumococcus and 37.8 per cent. to the streptococcus. The mortality in the first group was 18.1 per cent. and in the second 28 per cent. The table on page 480 gives summary of the bacteriologic studies and mortality figures of all our empyema cases. The cases classified as postpneumonia were admitted to the hospital for empyema too late in the course of the disease to determine accurately the form of pneumonia. Note the predominance of the hemolytic over the non-hemolytic streptococcus in the bronchopneumonias.

With complicating streptococcus infection the second form may occasionally occur with a lobar pneumonia.

Empyema complicating lobar pneumonia nearly always develops after the crisis, *i. e.*, during convalescence, as was the case in the first patient whose history I have given you. In spite of the purulent pleuritis the patient usually shows considerable improvement following the crisis. The fluid develops more gradually and, on the whole, with more suggestive signs than in the case of streptococcus empyema. It is at first thin

EMPYEMA

	Lobar pneumonia.		Broncho-pneumonia.		Post-pneumonia.		Total.		
	Number.	Died.	Number.	Died.	Number.	Died.	Number.	Died.	Mortality, per cent.
Pneumococcus Type I.....	14	2	..	..	6	1	20	3	15.0
Pneumococcus Type II.....	3	2	..	..	..	..	3	2	66.6
Pneumococcus Type III.....	1	..	..	..	1	..	2	..	....
Pneumococcus Type IV.....	5	..	..	..	1	1	6	1	16.6
Pneumococcus Type unknown	..	..	..	..	2	..	2	..	....
Streptococcus hemolyticus...	..	..	10	7	11	..	21	7	33.3
Streptococcus non-hemol....	..	..	1	..	3	..	4	..	....
Staphylococcus aureus.....	..	..	2	2	2	..	4	2	50.0
Undetermined.....	..	..	..	..	4	..	4	..	....
Total.....	23	4	13	9	30	2	66	15	22.7

and slightly cloudy, but soon becomes typical thick, creamy pus, occasionally showing a marked greenish tinge. Unless encapsulated the fluid forms in the lower portion of the pleural cavity even when the pneumonia is in the upper lobe. Other complications are not common and surgical treatment usually gives prompt relief of the symptoms of toxemia and the temperature drops to normal.

Streptococcus empyema, on the other hand, begins while the intrapulmonary process is still active, and its detection is, as a result, much more difficult. A particularly characteristic feature is its rapid development in contrast to the form just described. The onset has been observed as early as the fourth day of the pneumonia, and at this stage it may even obscure the signs of the process in the lungs. Within forty-eight hours the amount may reach 1 or 2 liters. Infection may take place on the unaffected side and bilateral involvement is not rare. The exudate is at first thin and of a dirty, cloudy character, but is less apt to assume a thick, creamy type than in the case of the pneumococcus infection. Complications in other parts of the body are frequent, the most common being infection of other cavities, septicemia, endocarditis, multiple abscesses in the lungs, encap-

sulated pus between the lobes or in the mediastinum, meningitis, and nephritis. An interesting feature occasionally seen in these cases is the development of a pneumothorax on the affected side, as occurred in Case II. The most probable explanation is that it results from the rupture of a small subpleural abscess of the lung, although, as in this case, it may be caused by injury to the lung tissue by the needle during paracentesis. The course of the empyema is prolonged and the mortality high, especially when due to the hemolytic type of streptococcus.

INCIDENCE OF EMPYEMA

A brief consideration of the frequency of empyema as a complication of pneumonia is of interest. Reliable statistics on this point are difficult to obtain. Musser and Norris collected 13,550 cases of pneumonia and found a complicating empyema in 2.2 per cent., and 5.1 per cent. among 973 autopsy cases. At the Johns Hopkins Hospital among 805 pneumonias empyema developed in 3.6 per cent. (McCrae). Empyema occurred in 6 per cent. of the 447 lobar pneumonia cases treated on the Pneumonia Service. The incidence shows a rather extreme variation from year to year and during epidemic periods. This was especially true of pneumonia as observed in the army camps. In the epidemic bronchopneumonia following measles and influenza the occurrence of empyema was often extremely high, nearly all fatal cases showing an infection of the pleura. Empyema developed in 16 per cent. of the lobar pneumonias and in 15 per cent. of the bronchopneumonias at Camp Devens (Gray). At Fort Riley 7.7 per cent. of 4000 cases of pneumonia were complicated by empyema (Stone). The figures for Camp Dodge are empyema with lobar pneumonia 11 per cent., and during the streptococcus pneumonia epidemic 34.8 per cent. (Miller). Cecil reports 50 per cent. empyema in a group of 118 bronchopneumonia cases (hemolytic streptococcus) at Camp Upton.

DIAGNOSIS OF EMPYEMA

The diagnosis of empyema is often extremely difficult. Gray's figures for Camp Devens emphasize this fact. Of 77 cases of empyema found at autopsy, 17 had not been recognized

clinically in spite of the fact that in 14 the amount of fluid equalled or exceded 250 c.c. A large percentage of cases will be overlooked if you pin your faith to physical signs alone, as they are notoriously untrustworthy. An "unresolved pneumonia" usually means empyema. The surest method is, remembering that empyema is the most common complication of pneumonia, to search diligently for it in every case of pneumonia until convalescence is well established. Remember that a large amount of fluid with resulting compression of the lung often gives the signs of consolidation, and even a massive effusion may, in consequence, be unsuspected. Remember also that in bronchopneumonia, especially when due to the hemolytic streptococcus, a large amount of fluid may develop in a comparatively few hours, and that it is in these cases that the signs are most frequently misleading.

The means at hand for diagnosis may be grouped under four headings: (1) the condition of the patient, (2) the physical examination of the chest, (3) *x*-rays of the thorax, and (4) exploratory puncture.

1. **The Condition of the Patient.**—The course of the temperature and pulse often gives more definite and direct evidence of the presence of a purulent fluid than the physical signs in the chest. The continued irregular fever as shown in the two charts of the cases under discussion is typical of pleural complications of this nature. After crisis in lobar pneumonia a temperature so slight as 1 or 2 degrees should always raise the suspicion of a focus somewhere in the pleural cavity. A disproportionately high pulse or embarrassment of respiration greater than can be explained by the process in the lungs is also suggestive. Pain of the pleural type and particularly when associated with friction should put one on his guard. Continued or increased prostration means probable involvement of the pleura in the absence of adequate cause in the lungs.

2. **Physical signs** may be characteristic or entirely deceptive, and must never be relied on exclusively when searching for fluid in the pleura. The sensation of resistance and loss of elasticity more than the tone with percussion over the affected area is an

important sign. Extension of the area of dulness at the base should be watched for. Muffling of the respiratory sounds points to accumulating fluid, but the breath sounds over fluid are in my experience as often intense and of bronchial quality as distant and obscure. Absent tactile fremitus is of far greater significance than changes in fremitus determined with the stethoscope. Localized tenderness over pus is not unusual in the infection with the hemolytic streptococcus. More obvious signs, such as immobility of one side of the chest, bulging interspaces, displacement of the heart or liver, merit no discussion, as they are late signs occurring only with large effusions.

3. ***x*-Rays** are indispensable to exact diagnosis in all doubtful cases, and in the hands of an expert roentgenologist the results are very reliable. Every pneumonia service should be equipped with a portable apparatus in order that repeated *x*-rays may be made with the patient in bed.

4. **Exploratory puncture** of the chest is entirely justifiable, as if properly done is without danger and causes the patient very little discomfort. As evidence of the absence of danger in aspiration, deliberate puncture of the consolidated lung for diagnosis has been done seventy-seven times on 71 patients in the Pneumonia Service, and in only 2 cases did empyema subsequently develop. Since this ratio is far below that for the incidence of empyema in our whole series of pneumonia it would seem fair to free the puncture of any causal relation to the 2 cases of empyema. We can never be certain of excluding fluid in certain instances without the use of the needle. One of rather generous caliber should be selected, as thick pus with fibrin will often plug a small one. My advice is to "needle the chest" early and often when pus is suspected. When all other evidence of an effusion have been sought for, paracentesis is the final and most reliable means of settling the question.

TREATMENT OF EMPYEMA

I shall not attempt a discussion of the surgical technic or the various methods of effecting drainage of the infected pleura. Such subjects belong to the surgical rather than the medical

clinic. There are a few considerations, however, affecting the time for operative interference and therapy other than surgical which I should like to bring to your attention.

On general principles one must be guided largely by the condition of the patient and the type of organism present in deciding when to begin surgical treatment. The early experience with the empyemas in several of the United States Army camps proved the folly of attempting to operate all empyemas early. It would be an easy matter to quote figures in evidence of the appalling mortality which resulted. In several instances the mortality was as high as 50 per cent. The patient's chances of recovery are materially increased if operation can be deferred until the process in the lung has passed the active stage and there has been sufficient time for improvement in the general condition. Fortunately, as mentioned earlier, the infection of the pleura in lobar pneumonia usually takes place relatively late, that is, the symptoms of the empyema appear during early convalescence from the pneumonia. At this stage the individual is in much better condition to stand operation than earlier. To a limited degree it is often justifiable to delay surgical treatment for a few days or even a week in the case of pneumococcus empyema.

In the hemolytic streptococcus empyemas following the bronchopneumonia of measles or influenza, on the other hand, the situation is a serious one. The complicating empyema not only develops suddenly, but usually coincidently with the intrapulmonary process. These patients are extremely toxic and in no condition to withstand an operation. After simple aspiration surprising improvement in the patient's general condition often results. By repeated tappings at intervals of one to several days the operation can with but few exceptions be postponed until such time as the resistance of the individual is more favorable. The experience of a single large clinic is sufficient to illustrate the point. Major Stone reports the results in 310 cases of empyema treated at Camp Riley; 35 cases recovered with repeated aspirations alone; 85 (pneumococcus 19, streptococcus 52) were treated by early operation (October 20, 1917 to January 21, 1918) with a mortality of 61.2 per cent.; 96 (streptococcus 69,

pneumococcus 24) were aspirated early and operated late (January 12 to August 10, 1918), with a mortality of 15.6 per cent.; 94 (streptococcus 59, pneumococcus 25) were aspirated early and operated late (October 18, 1918 to February 14, 1919), with a mortality of 9.5 per cent.

The routine on the Pneumonia Service is as follows: In the case of pneumococcus empyema, when the exudate has become frank pus, operation has been done at once, provided the patient has reached the point in convalescence from the pneumonia where his general resistance may be classed as good. In less favorable cases operation has been delayed often for several days. With the streptococcus empyemas aspiration has been done early and repeated every two to four days until the acute and critical stage of the lung condition had passed. The time for operation is decided largely on the basis of the general condition of the patient. After operation the patient remains on the Pneumonia Service, and in consequence has the advantage of medical as well as surgical supervision.

Far too little attention is given in the average clinic to the general hygiene and nutrition of the empyema case. Improvement of the general resistance of the patient is of quite as much importance as the local surgical treatment of the infected pleura. Whenever possible open-air treatment should be carried out. If left to follow the dictates of his own appetite the patient very seldom takes an adequate amount of food. Rapid and marked emaciation is the rule. The Empyema Commission appointed by the Surgeon General during the war conducted metabolism studies on streptococcus empyema cases and found a marked increase in the nitrogen elimination indicating an enormously increased protein metabolism. The Commission reports successful results in checking the emaciation by the use of a special dietary having a fuel value of from 3000 to 3500 calories.

MORTALITY IN EMPYEMA

The mortality in empyema is at best high. The reasons for the high death-rate are not far to seek. Empyema is a complication in a serious acute infection which itself has a high rate of

mortality. Very often the infection of the pleura is associated with septic foci elsewhere, *e. g.*, multiple abscesses in the lungs, pericarditis, peritonitis, encapsulated pus in the mediastinum or between the lobes of the lungs, or even a septicemia. This is especially true in the hemolytic streptococcus infections. Factors of no small importance affecting the death-rate are the time of operation and the method employed. Skilful post-operative care will save many. For unoperated cases the percentage of deaths varies considerably, but it seems a safe statement that from two-thirds to three-fourths of such cases die.

The general mortality for all empyemas treated at the Boston City Hospital for the years 1915–1918 was 21.6 per cent. From February 6, 1919 to April 30, 1920, 66 cases of empyema were treated on the Pneumonia Service. The mortality among the 55 operated cases was 20 per cent. The most favorable statistics are those of Whittemore. He reports 100 operation cases, with a mortality of only 6 per cent. Extremely variable and usually high death-rates have been reported from the army camps. As these cases for the most part occurred during severe epidemics of bronchopneumonia (streptococcus) the figures probably do not indicate the results usually to be expected.

The period of the pneumonia at which the empyema develops bears a definite relation to the chances of recovery. Gray (Camp Devens) found that 46 per cent. of those developing empyema during the first week of their pneumonia died, 26 per cent. of those in the second week, and only 7 per cent. of those later than two weeks. These results illustrate very well the fact that the earlier the empyema occurs in the course of a pneumonia, the worse the prognosis.

The prognosis of empyema with lobar pneumonia is much more favorable than with bronchopneumonia. In our cases the mortality was 17 per cent. in the first and 69 per cent. in the second group.

Convalescence after operation in empyema is a tedious affair, usually covering a period of two or three months. The duration of treatment after operation in Case I was fifty-eight days; in Case II, eighty days.

CLINIC OF DR. FRANKLIN W. WHITE

BOSTON CITY HOSPITAL

THE MODERN EXAMINATION OF THE STOMACH

WE are in a transition stage of examination of the stomach. Old methods are being discarded and new methods used. Some men are saying good-bye to test breakfasts and stomach contents and blood tests and relying entirely on the *x*-ray examination. Some men are trying to use the Rehfuss tube and getting a lot of data which they find hard to interpret.

This paper is one of comment on the various up-to-date methods and a consideration of which to use and which to discard, which are important and which are unimportant, the relations of one test to another, and how to use the best ones to make a diagnosis.

A careful history is still one of our most valuable means of diagnosis, and after taking it, a provisional diagnosis can usually be made, which will be confirmed or modified by other methods of examination.

It is always very important in our examination to distinguish between organic and functional diseases of the stomach because organic disease requires local treatment, while functional secondary or reflex digestive disorders due to diseases of the lungs, heart, kidneys, liver, central nervous system, etc., require chiefly treatment of the primary cause.

Some of the older methods of examination, such as inspection, palpation, and percussion of the abdomen, are quite imperfect, but they are easy and require no apparatus, and give us some very valuable data, such as local tenderness, palpable tumor, visible peristalsis, etc. Changes in secretion were formerly overestimated as a means of diagnosis of surgical lesions and they have had to give way here to the Roentgen examination, but in the great group of functional cases, which includes nearly nine-

tenths of all the patients seen, a knowledge of the gastric and intestinal functions is one of our chief aids in diagnosis and treatment.

The Roentgen examination has developed very rapidly in the last few years and has become our most valuable single method of examination, but must not on that account be allowed to overshadow all other methods. The bad practice is growing of sending poorly studied digestive cases to the radiologist and accepting his word as final. This is a mistake. Roentgen evidence should be a part of clinical evidence and considered with it. Furthermore, the Roentgen examination may be negative, but the patient far from well, owing to conditions easily found by other methods, for example: a patient with achylia gastrica and persistent diarrhea after a complete Roentgen examination gets a negative report, while a single tube test gives the diagnosis.

The careful history, the general examination of the patient, the functional tests of the stomach and bowel, all taken with the Roentgen evidence, give our most accurate diagnosis and surest basis for treatment.

History.—A careful and thorough history is one of the greatest aids in diagnosis and treatment, and is well worth all the time it takes.

In the early stages of many diseases of the stomach, like disturbances of internal secretion, the physical examination shows nothing abnormal and we have to depend almost entirely on the history alone for our diagnosis.

Hyperacidity or "hunger-pain" is always a striking symptom. The frequency of hyperacidity in ulcer has tempted so great a surgeon as Moynihan to say that "hyperacidity is ulcer." This, of course, is not true, yet he did us a great service. In severe chronic "hunger-pain" we no longer make the diagnosis of simple hyperacidity and let the matter rest there; we look further in every case to see if we can find an organic basis for this symptom, and in the severe persistent cases we usually do find an organic basis. This is a chronic ulcer in about two-thirds of the cases of severe hyperacidity, and either a chronic appendix or a chronic gall-bladder in the other third.

It is a waste of time to try and locate an ulcer by the symptoms alone. If we watch the stomach with the fluoroscope we find that the pylorus usually opens within a few minutes and food is discharged into the duodenum. In other words, within a short time after food is taken an ulcer, whether in the stomach or in the duodenum, is equally bathed in food, the irritation of acid occurs just as promptly in one as the other; both are equally stirred up by peristaltic movement and spasm. It is far more satisfactory to look directly at the ulcer with the Roentgen ray and *see where it is*, than to trust to symptoms to locate it.

We must always remember that there are two distinct classes of patients with "stomach symptoms," those with actual disease of the stomach, such as cancer, ulcer, catarrh, etc., and those in which the symptoms are the expression of disease elsewhere in the lungs, heart, kidneys, spinal cord, etc. In doubtful cases we must look carefully for other symptoms, such as tuberculosis, nephritis, heart disease, tabes, etc. We must also remember that *reflex symptoms and pain are the rule, not the exception, in the abdomen*, and we must try to distinguish symptoms due to the gall-bladder and the appendix from those due to actual gastric disease.

There are serious mistakes in history taking for diagnosis. One is to overlook the important psychic or emotional causes of indigestion, and the other to conclude that because the patient is a pronounced neurasthenic that there is nothing else wrong with her. For example, we ask our patient about infections and diet, but we don't ask her if she is happy with her husband or her mother-in-law. I constantly examine middle-aged women sent me because cancer is suspected, and find that the husband is flirting with another woman, or the daughter is unruly, or all the servants have left.

On the other hand, nervous symptoms, such as headaches, insomnia, worry, excitability, may be so obvious and many that the doctor rashly concludes that the trouble is "just nerves" and nothing else, which may be a very serious mistake. Let me give you one or two illustrations:

The first case, Mrs. H. E. D., aged fifty-eight, has always been

a nervous woman. She had a nervous breakdown fourteen years ago, which lasted two years. She fell downstairs four years ago, and the nervous shock of this lasted for a year. Her daughter has recently had eclampsia and recovered only after five or six weeks' severe illness. The nerve strain of this was very trying to our patient. She had for five weeks epigastric distress at once after meals, lasting from one-half to one hour, with "gas" and occasional regurgitation of a little stomach contents, also nausea an hour and a half after meals, often relieved by food; no vomiting. She has lost her appetite, become a little constipated, and has insomnia, and has lost about 15 pounds. She has peculiar ideas about food. "Can't eat bread," but can eat cereals, cake, etc.

Physical examination shows a well-developed, plump, soft, flabby, nervous woman, with good color. Nothing important was found in heart, lungs, abdomen, or rectum; reflexes, blood-pressure, urine, and blood were normal. The stomach contents, after a test breakfast, was normal in amount, with low normal secretion (free acid 23, total acid 26); no mucus. Blood positive with benzidin.

A very careful and thorough *x*-ray examination of stomach, bowel, and gall-bladder showed nothing abnormal.

The diagnosis of neurasthenia and a gastric neurosis was made on the basis of the previous and present nervous condition, and the negative physical examination. The suspicion of early cancer could not be verified.

The patient improved considerably on careful diet and medical treatment. The digestive symptoms lessened a great deal; the sleep became quite good and she became stronger. The stools were repeatedly tested on a meat-free diet, and *always gave a positive test for blood.* This important finding led to a second examination of the stomach and bowels, including a barium enema six weeks later, at which nothing definite was found. An exploratory operation was advised, but was refused at first by family and patient, but was finally done five months after the original examination. At that time a large inoperable cancer of the stomach was found involving the median posterior wall and

fundus of the stomach. When the *x*-ray plates taken three months before were looked over again a small linear rigid area ½ inch long in the middle of the lesser curvature was found constant in all the plates. This had been overlooked, but was evidently a very small lesser curvature cancer area.

This case illustrated several things: first, the tendency to minimize the early symptoms of a serious disease in a very nervous woman, and to attribute them to neurasthenia. Second, the difficulties in diagnosis of early cancer by all methods, including the *x*-ray, in flabby, weak, elderly people, with poor peristalsis and poor filling out of the stomach. Third, the value of the blood test in controlling the *x*-ray examination and keeping the case on the suspicious list in spite of negative *x*-ray reports.

The second case, Mr. F. G. C., is a very hard working factory manager, a typical overstrenuous business man who always rushes his work. His sleep is poor and restless, and rarely over three or four hours a night for the last five years. There are no bad habits or important previous illness. He has had intermittent distress in the epigastrium at once after meals, relieved by two or three soda mints, for the last eight or ten years.

In the last seven months he has had daily distress or gnawing in the epigastrium four or five hours after meals; with food relief at once, also slight distress at 12 to 2 A. M., with food relief. No nausea, regurgitation, vomiting, or loss of weight. He has eaten everything. He is always costive and uses mineral oil and cascara.

Physical examination shows a heavy muscular man; weight 200 pounds. Good color; nervous. Nothing abnormal was found in heart, lungs, or abdomen. Reflexes, urine, and blood were normal. Stomach contents after test breakfast showed complete gastric achylia (amount 95 c.c., free HCl 0, total acid 6; no mucus or blood). *x*-Ray examination showed nothing abnormal in stomach or bowels. The diagnosis of achylia gastrica of nervous origin was made and treatment given.

The patient did not do well, and a month or two later showed a constant positive test for blood in the feces. A second *x*-ray examination, three months later, showed a small rigid area about

the middle of the lesser curvature and a little to the posterior side, in which there was complete absence of peristalsis. There was a moderate six-hour residue. A diagnosis was made of "a lesion" of the lesser curvature of the stomach. It was impossible to say from the *x*-ray examination whether this was due to ulcer or cancer.

He was operated upon within a week, and a large inoperable cancer found involving the upper half of the stomach posteriorly.

This case, like the previous one, shows a mistaken early diagnosis of a gastric neurosis, based on the patient's nervous condition and negative physical examination, an early cancer in the upper posterior part of the stomach, missed by all methods of examination, and the value of a positive blood test in the feces, in spite of a negative *x*-ray report.

In the diagnosis of cancer we must never wait for marked symptoms. We must get the text-book pictures of cancer entirely out of our head. If we wait for coffee-ground vomit, obstruction, lack of free HCl, palpable tumor, emaciation, etc., we have an absolutely hopeless case.

The question is often raised whether primary cancer of the stomach is *ever* diagnosed early. A small cancer very rarely is, unless it is just at the pylorus.

I want to emphasize, however, that the *cancer developing on ulcer is and should be regularly diagnosed early*, not by the symptoms, but by the modern methods of examination which I will describe.

The only way to find early operable cancer is to examine promptly and thoroughly all patients of cancer age with digestive symptoms.

I have passed rather briefly over the use of symptoms in diagnosis, not because it is unimportant—far from it—but because there is little new. Most of the recent progress in the diagnosis of diseases of the stomach has been made by improving the methods of physical examination.

The following general outline of questions is suggested:

Family History.—Especially alcoholism, nervous diseases, tuberculosis, obesity, malnutrition, diabetes, cancer, gout, arthritis.

Previous History.—Especially typhoid, dysentery, appendicitis, jaundice, abdominal operation, tuberculosis, syphilis, infections of teeth, sinuses, tonsils, nervous breakdown, migraine, headaches, insomnia.

Occupation and Habits.—Tea, coffee, tobacco, alcohol, eating habits, sleep, exercise, work, temperament, home life, social life.

Present Illness.—Duration, onset, abrupt or gradual.

- Attacks or continuous
  - Number,
  - Character,
  - Duration,
  - Interval.
- Dysphagia
- Pain and distress
  - Intensity and kind,
  - Where (local, diffuse, radiation),
  - Regularity.
  - Relation to food
    - At once,
    - One to two hours after,
    - Two to five hours after.
  - What stops, relieves or increases
    - Food,
    - Alkalies,
    - Vomiting,
    - Belching,
    - Passing gas or feces.
- Gas
  - Amount,
  - Belching (taste),
  - In bowel,
  - Passed (odor).
- Vomiting
  - Frequency,
  - When, relation to meals,
  - Quantity.
  - Kind
    - Food,
    - Sour, bitter, or foul,
    - Blood (bright, dark, or coffee grounds),
    - Mucus.
  - Regurgitation (gas, liquid, food).
- Bowels
  - Constipation,
  - Laxative used,
  - Gas,
  - Painful defecation,
  - Feces (consistency, color, odor, shape, mucus, blood, undigested food, worms).

General condition.....	Loss of weight (amount, rapidity), Strength, Appetite, thirst, bad taste, jaundice, Ability to work, Nervousness (dulness, sleep, headache, worry, etc.).
Diet.....	General, Restricted, how long? Rational.

Physical Examination.—Before studying the digestive apparatus, the patient should have a complete physical examination. It is very important that digestive symptoms due to tuberculosis, nephritis, tabes, etc., shall be traced to their actual source. We especially examine teeth, tonsils, and sinuses for sources of infection of ulcer, gall-bladder, and appendix.

Some of the older methods of examining the stomach and bowel are very imperfect and the results vague, but they are easy and require no apparatus and give us some very valuable data; such as local tenderness, palpable tumor, visible peristalsis, etc.

Palpation is an important method, but we must know what to expect and what not to expect.

It is striking how little we can *feel* in comparison with what we *see* with the Roentgen ray. In reviewing a large number of ulcer cases I find it is very rarely that I am able to palpate the ulcer. This is easily understood when we remember that the lesser curvature and the duodenum are the favorite sites with ulcer, and these are so frequently hidden under the edge of the ribs or liver that they are entirely out of reach when the patient lies on his back. After having seen the ulcer with the Roentgen ray I have frequently returned to the patient in bed and absolutely failed to get any clear evidence of its presence by palpation. I have just said that we must not wait for a palpable tumor in order to diagnose cancer.

Tenderness in the epigastrium is so common that it has little diagnostic value unless it is sharply localized, then it suggests ulcer or cancer (or the pyloric spasm found quite often in disease of the duodenum, gall-bladder, or appendix). Tender-

ness over the tenth to the twelfth ribs, behind on the left side, is occasionally found in gastric ulcer, but is a very unimportant sign in diagnosis.

Percussion of the outline of the stomach is a method of little value. If we percuss the stomach without inflating it, the area of resonance is about as likely to be the splenic flexure as the stomach, and if we percuss the stomach after moderate inflation through the stomach-tube, or by split Seidlitz powder with the patient on the back, we percuss the area of the gas bubble which rises up under the abdominal wall in the middle of the stomach which gives us very little idea of its actual outline. It is a very crude method and hardly worth the time it takes.

The **test of capacity** of the stomach, by pouring in liquid until the patient cries enough, is of little value. The capacity of the stomach depends entirely upon the tone of its muscles. The normal stomach embraces its contents closely, no matter how small they are, and the amount which can be poured in without discomfort depends almost entirely upon tone and sensitiveness, and varies greatly in different people with stomachs of the same size. We have other better ways of detecting a dilated or atonic stomach.

Some other methods of examination, such as the gastrodiaphane, desmoid capsule, glutoid capsule, etc., are obsolete and will not be described.

Gastric Contents.—The use of the stomach-tube to test the secretion and emptying of the stomach has received a great set back since the free use of the Roentgen ray. Many doctors are asking Why should we bother with the stomach contents any more; the tests are not very important; why not trust to the *x*-ray alone? Why have so many doctors lost their interest in functional tests of the stomach?

Let me tell you. Because they expected too much and have been disappointed. They expected to diagnose ulcer by the stomach contents alone and they find that most ulcers if not obstructive have average secretion.

They expected to diagnose cancer in the same way, and find

that the secretion often does not change much until the cancer is well developed and hopeless.

They find that the *x*-ray examination is far more definite in both ulcer and cancer, and they decide that they will not bother with test-meals and gastric analyses any more. They will simply divide all stomach cases into surgical and non-surgical on the basis of an *x*-ray examination. In the vast majority of cases they will simply accept the rontgenologist's report of "no pathology."

This is a mistake. There is no reason to give up the stomach-tube because we have the Roentgen ray. Tube tests are simple and cheap and give us valuable information. They are the only means of testing gastric secretion, and if secretion is either high or low we want to know it. They also tell us whether or not the stomach empties properly and about bleeding.

Furthermore, cancers and ulcers are only a small fraction, probably less than one-tenth, of the cases we see with "stomach trouble." This great group, containing over nine-tenths of all dyspeptics, will have to be treated in some way. It is not enough to simply lump together every sort of high secretion case, achylia, catarrh, neurosis, etc., in one great class and label them "non-surgical" or "no pathology" and treat them all alike. They must be classified largely on the basis of functional tests and proper *individual* treatment given. The physician who will not take the trouble to do this is not fit to treat them.

When we test gastric secretion we do not simply find the change caused by the disease present. We get *two things at least*—the *personal individual reaction* of that stomach to the test-meal and also the *disease changes*. Often we are *typing the individual* even more than the disease.

This *individual reaction* to a test breakfast is very variable, for example, it is usually much lower in the thin, tired woman than in the vigorous tobacco-using man of the same age, when both are *well*.

One of the chief reasons for testing the gastric secretion in stomach cases, such as neurosis or ulcer, is to find out whether they are high acid or low acid cases, not so much for *diagnosis*

as for *treatment*. We avoid in this way the overuse of alkalies in low acid ulcers and we also avoid dosing the patient with acid, pepsin, and other ferments when he already has an abundance of these things. In other words, we can use our drugs for a definite need instead of giving them hit or miss as is so commonly done.

Test Breakfast.—The Ewald test breakfast is the standard—1½ thick slices of stale bread (60 grams) with 1½ glasses (350 c.c.) of water. Many modifications of this standard meal have been proposed, even down to plain water. They have no advantages which compensate for the loss of the *standard quality*, the opportunity to compare results with immense numbers of other results all obtained in the same way.

The Ewald meal is primarily a test of secretion, but gives us some information also about the emptying time of the stomach. A simple and satisfactory test of gastric motility is to give the patient a handful of seedless raisins or a saucer of stewed prunes twelve hours before the test breakfast (say at 9 P. M. the evening before). If some fruit residue is found mixed with the test breakfast next morning we have an important disturbance of gastric motility. Special motor meals are largely replaced at present by the six-hour Roentgen examination.

The Stomach-tube.—The whole secret of using this test freely is to make it easy for the patient. This is done in three ways: by spraying the throat, by using a small stomach-tube, and by using an aspirator. No one of these things is absolutely essential, but each helps greatly to make the examination short and easy.

The throat spray of novocain (2 to 4 per cent.) is just as useful as it is in minor surgery, and takes away most (not all) of the gagging and throat reflexes.

A small tube is as good as a large one. We do not need a hose-pipe to get stomach contents; witness the growing use of the Rehfuss tube, no larger than a quill.

The aspirator, the Ewald bulb, which has been in use for twenty years or more, is too much neglected. It never does any harm and gets the contents (if there is any) in a half-minute or

less where the *expression* of contents is often tedious and trying for the patient.

We must emphasize the *simplicity* of modern routine examination of gastric contents. After all frills and unnecessary apparatus are pruned away, it will take no longer than a routine examination of urine for reaction, gravity, albumin, sugar, and sediment. This includes testing the stomach contents for amount of mucus, acidity, and blood, and takes not over five or ten minutes. Tests for ferments are only done rarely, and the microscopic sediment rarely shows us anything we have not already learned in other ways.

This simplicity is shown by the fact that the average good stenographer or secretary, with very little medical or chemical training, can be shown the routine tests of stomach contents, just mentioned, in an hour's lesson and continue to do them accurately with very little supervision.

Rehfuss Test.—*The Rehfuss fractional examination of stomach contents* is very valuable for research upon the gastric functions, but has reached thus far only limited use as a purely diagnostic method. It has given us a large amount of information about the complicated changes which occur in the normal stomach during the entire process of digestion of a meal, the types and phases of normal digestion, the digestive and interdigestive cycles, the normal gastric response to all kinds of food and drugs; and charting the results gives interesting curves both of gastric secretion and motility.

Recent work with the Rehfuss tube has shown that the *normal limits of secretion are very much wider* than formerly supposed. The *average* normal figures for free HCl are, namely, 20 to 40, but we often find 60, 70, and even 80 in perfectly healthy persons with a total acidity of 100, 150, and even 200. In short, there is no figure found in disease which is not duplicated in normal persons, and the question is fairly raised, Is there such a thing as true hyperacidity? (an acid secretion higher than normal). We must think of "hyperacidity" as a group of symptoms—hunger-pain, distress, burning, gas, eructations, etc.—relieved by food or soda (and associated with painful peristalsis

or spasm, or esophageal regurgitation, or "low tolerance for acid") rather than an actual increased acid secretion *above* the normal.

In disease the Rehfuss tube shows changes in the speed of secretion and digestion all the way from total suppression to exaggeration of every phase, the alterations of digestion caused by the addition of blood, pus and mucus, and by regurgitation of duodenal contents, the relation of pain to secretion, etc. The curves of secretion are characteristic of *disease groups* rather than of individual diseases, for example, we have the high short curves of gastric and duodenal ulcer, disease of the gall-bladder, appendix, etc., and the low slow curve found in chronic gastritis, tuberculosis, anemia, nephritis, and gastric cancer.

The method is elaborate (and complete) by comparison with the usual tube test, and for that reason is not used commonly in *routine* chemical examination at present, but is reserved for selected cases of particular interest or difficulty, for example, in distinguishing true achylia from delayed secretion. In true achylia, gastric secretion is absent throughout the whole process of digestion. In spurious achylias at the end of one hour there may be no free acid, and yet quite an abundance after this period.

The Rehfuss method has many advantages over the single tube test one hour after a test breakfast, which only indicates one phase in a constantly changing cycle, which is not always the high point of secretion and which tells us nothing about the phases preceding and following it. On the other hand, gastric secretion has limited diagnostic value, and the simplest method, the single tube test, even if it is only approximate, is usually preferred. The actual *clinical* value of the Rehfuss method will be better understood when it has had a wider use.

We may combine the simplicity of a simple tube test with many of the advantages of the fractional method by passing the Rehfuss tube forty-five or fifty minutes after the test breakfast and aspirating a few cubic centimeters of stomach contents and testing with Topfer's reagent; if abundant free acid is present the stomach contents may *all* be withdrawn and examined and

the test closed. If little or no free HCl is found the tube may be left in place and small samples aspirated every fifteen or twenty minutes until free acid (or more free acid) is found or the stomach is empty. In this way normal secretion cases are quickly disposed of, while delayed or low secretion cases are promptly and thoroughly followed up.

Blood Tests.—Do not forget that you have at your disposal a very simple test to discover some of the most serious gastro-intestinal diseases and check up negative results of other methods, namely, the blood tests of feces or gastric contents. The first *x*-ray examination may miss a cancer of the colon or fundus or esophagus, but a constant blood test marks the case as dangerous and requires explanation, and should lead to a second or repeated examination, until the case is clear. This checking up of one method of examination by another, *e. g.*, the *x*-ray by blood tests, is very important. I have seen its value demonstrated many times.

Personally, I much prefer the benzidin to the guaiac test on account of its somewhat greater delicacy and easier technic.

The simplest blood test for the practitioner is the benzidin tablet of Dudley Roberts, put up by Squibb & Sons. It requires no apparatus but a clean saucer. One tablet is placed in a saucer with enough stomach contents to wet the tablet thoroughly, but not to cover it. Then a drop or two of glacial acetic acid is dropped upon the tablet; if blood is present the tablet turns greenish blue. The same test can be used with feces, thinned with a little water.

Blood tests should not be relied on to diagnose ulcer, since we only get a positive result in about one-third of the cases. They do help us to judge the activity of an ulcer and also to rule out cancer. The latter is far more likely to give a positive test and give it constantly.

The **string test** consists in having the patient swallow a silk thread about a yard long, leaving it in the stomach over night and pulling it out in the morning. A brownish stain on this thread "indicates ulcer." This test has all the advantage of ease and simplicity. Is it, therefore, a good practitioner's test? No,

decidedly no, because in my experience it is *usually negative* when ulcer is present. It is therefore misleading and helps us to overlook an important disease. It ought not to be used to diagnose the presence of ulceration, only perhaps to show the activity of an ulcer, and even here it has doubtful value. Frank Smithies, of Chicago, found it positive in only 7 out of 318 gastric ulcers verified at operation. I have given it a thorough trial, though I have not had the patience to use it in nearly so many cases, and have given it up.

The Roentgen Examination.—The Roentgen method can tell us so much about the digestive tract that we must all become familiar with Roentgen pictures and their interpretation just as the medical students are doing today. The method has given us entirely new ideas of the shape and position of the living stomach and the changes in outline and function which come with disease. It is the most valuable *single* method of examination, but it must not on that account be allowed to completely overshadow the others.

The different kinds of Roentgen evidence have very far from equal value; an esophageal pouch, old cancer of the stomach, and calcified gall-stone are in a very different class from early cancer, cholesterin stone, and simple adhesions. It is also very hard at times to tell personal peculiarities from signs of disease.

As a rule both the fluoroscope and plates should be used; the two methods supplement each other, and something is lost if either is omitted. The plate gives us fine detail which we may miss in looking at the moving stomach. The fluoroscope which enables us to watch the stomach in action gives us a multitude of successive pictures which could only be duplicated by scores of plates; we watch peristalsis, we can push the stomach about to test for adhesions or fixation, we can locate a tender point. In general we can say that least mistakes are made in those laboratories where both methods are used. The proper interpretation of fluoroscopic shadows or plates requires large experience and good judgment and conservatism. It is wise to control every finding by re-examination. To subject a patient to needless operation on slight Roentgen evidence alone is a serious matter.

How Often to Use it.—It is difficult to decide just how often the Roentgen method should be used. Time and expense, of course, must be considered. On the other hand, we must not miss a cancer or ulcer or treat any chronic case without trying to find out what is wrong. Cases may be wrongly diagnosed by careful and expert physicians and undergo months of useless treatment, when the right diagnosis could be made by a single *x*-ray examination. On the other hand, we must not be disappointed if the *x*-ray occasionally shows us nothing at all in very troublesome cases. This frequently happens in functional cases with most trying symptoms of long duration, but this negative examination may prove very valuable to a nervous person by ruling out serious organic disease.

We should at least use the Roentgen method:

In all patients in whom a serious disease, like cancer or ulcer, is suspected.

In all patients of cancer age with definite digestive symptoms.

In most patients with digestive trouble of long standing, especially if not relieved by careful treatment.

In some neurotic persons largely for the encouragement of finding nothing serious.

To replace exploratory laparotomy as far as seems reasonable.

Limitations of the Method.—It is no discredit to any method of examination to say that it has limitations, and the failings of the *x*-ray method must be recognized, just like the limitations of palpation of the stomach or gastric chemistry or blood tests, already spoken of. We do not expect to palpate a duodenal ulcer, and we must not always expect to find a cancer in a partly filled esophagus or colon or stomach fundus with the *x*-ray.

In addition, the *x*-ray illustrations and description usually come from typical beautifully clear-cut cases like the typical clinical pictures of typhoid or phthisis or cancer. We should rarely fail in our diagnosis if all the cases were clear cut and typical. The typical case does not show our difficulties. We have the poorly filled atonic stomachs in flabby, weak, elderly people, without much peristalsis in which a small cancer may be missed. We have the difficult fundus cancers which are fortunately rare.

We have some poor plates in heavy patients and in stupid or nervous patients, who will not hold their breath, and we have patients with abdominal fluid, which fogs the plate.

There are real mechanical difficulties to be overcome in *x*-ray work, and the routine plate is not always the typical beautiful demonstration plate that is picked out for illustrations and exhibitions.

There is no difficulty in finding a *large* cancer of the stomach, and we may note in passing that in the large series reported by several authors a few years ago with 95 per cent. correct diagnosis, *about 70 per cent. had a palpable tumor.* In the diagnosis of *early primary* cancer of the stomach (the only kind we are really interested in) we must admit that the *x*-ray, like every other present clinical method, usually proves a failure. Men of large experience now and then report an isolated case or two, that is all.

In the partly filled stomach the small defect of a non-obstructive cancer may be easily missed, and then, of course, we have errors of interpretation—the *partial filling* of a stomach or colon may be mistaken for a cancer defect or the spastic and trying duodenum which will not fill even after a long examination, may be rashly called an ulcer.

Re-examinations and Follow-up System.—This brings me to the need of re-examinations and a good follow-up system, of several months, at least, in many stomach cases both to avoid mistakes in diagnosis and to judge the results of treatment.

A single *x*-ray examination is often relied upon, as it used to be with test-meals and blood tests. This is easily understood, partly on account of the time and expense involved on *x*-ray work, and partly because both patient and doctor often expect that with this new and truly remarkable method one good examination is enough. Of course, we never expect one clinical examination to be enough in chronic nephritis or suspected tuberculosis, and the same thing is often true of *x*-ray examination of the stomach. This re-examination to decide some important or *special point* about ulcer or possible cancer of the stomach need not be long or expensive, and is often very valuable indeed.

It is often a wise plan to verify an important pathologic finding by a second examination.

No man really deserves the name of physician who does not systematically follow up a case of diabetes, and I am inclined to think that the same is true in chronic ulcer of the stomach and duodenum, and it certainly is true in suspected cancer of the stomach. Follow ups are somewhat tedious, and may require a secretary, but they can be systematized and are well worth while, and it is a great comfort to the doctor to know that his chronic cases are getting well, and it is just as great a satisfaction to know that by his care and patience he has avoided making a wrong diagnosis, and has not called a case "nerves" when it is really cancer, and has perhaps saved the patient from endless suffering or death.

We often have a fine chance to control one type of examination with another, such as a blood test and an *x*-ray examination. If we suspect cancer we must not be satisfied with a negative *x*-ray report, especially if the patient shows a positive blood test constantly in the stools on a meat-free diet and in the absence of piles. We must settle the discrepancy between tests by re-examinations and a good follow up, and get at the truth of the matter. I have repeatedly seen serious mistakes avoided in this way.

The Normal Stomach.—What is a normal stomach? We are accustomed to think that "A stomach is a stomach whoever has it" and expect it to be just so long and just so wide and just so high and in just such a place, but on *x*-ray examination we find that there is great variety in normal stomachs—they correspond to the shape and build, *the habitus* of their owners—some are oblique, some hook shaped and low, some transverse and high, some globular, some narrow, and it is always striking to find that the smallest and weakest people seem to have the largest stomach (through lack of tone) and the largest and strongest people have the smallest (most compact and muscular) stomachs. The important thing to remember is the immense variety of normal stomachs; they are like peoples' faces—we have general types, but we can hardly find two which look just

alike—and we must always be on our guard not to interpret *personal peculiarities* of shape or position or tone or peristalsis as signs of disease.

An *x*-ray report of ptosis has definite value, but we must not lay too much stress on it; a little experience will usually tell us where a person's stomach will be found. To say that a man has ptosis is much like saying that he is long and thin and has flabby muscles and poor posture, which does not require much apparatus or an expensive examination, but merely the use of the eye. Never forget that the *functions* of a stomach, its emptying time and its secretion, are more important that its mere *position*. Many of us do perfectly well with a stomach that is lower down than the average.

The Emptying Time of the Stomach.—This can be decided either by the stomach-tube or the *x*-ray. We all know that disturbance of this important function is one of our most valuable signs of disease. There is no need to emphasize this. When the doctor gets an *x*-ray report that the stomach does not empty in six hours after a barium meal he begins to think of an obstructed pylorus, and possible gastric enterostomy. What I want especially to emphasize is the *frequency* with which delayed emptying occurs in non-obstructive cases and the many factors which may cause it, and the *large normal variation* in emptying time.

Barium meals have varied much, but at present 3 ounces of barium sulphate with 12 to 16 ounces of cereal gruel or fermented milk may be considered standard, and the average emptying time from three to six hours. The two great factors which control it are the muscular tone of the stomach and the condition of the pylorus. On account of the first factor, the emptying time corresponds to the habitus or body form—rapid in the broad and slow in the slender person. The emptying time in the very strong person is three to four hours, in the strong four to five hours, in the average five to six hours, and in the weak person six to seven hours. There are many normals, what is slow for one is not slow for another.

The six-hour barium meal residue is a more delicate test of

gastric function than the twelve-hour test with the stomach-tube, and we naturally expect the former to show more positive results in functional cases.

If we use the six-hour barium examination as a test of function we preferably allow no food in the interval. The radiologist is usually more interested in finding an actual lesion than in studying function, and is sometimes careless about the latter. I have repeatedly seen cases of ulcer in which a report of a good-sized six-hour residue was made, and found that the patient took his barium meal at 10 A. M., took a hearty lunch at 1 P. M., and went for the six-hour examination at 4 P. M. The lunch held back some barium in the stomach and gave us a striking "six-hour residue."

Other factors which delay the emptying of the stomach by lessening its muscular tone are nervous depression, headaches, especially of the migraine type, nausea, fatigue, and old age. Mills, of St. Louis, found in a very large series of cases that while peptic ulcers, duodenal and gastric, showed six-hour barium residues in about 50 to 80 per cent. of the cases, and 32 per cent. of the gall-bladder cases and 15 per cent. of the appendix cases gave the same result partly through spasm and partly through adhesions, he *also found* a six-hour residue in 28 per cent. of the cases of atony and ptosis and in 64 per cent. of the cases of migraine, and in about 50 per cent. of all elderly people of seventy or over. This shows clearly enough that an *x*-ray report of a six-hour residue must make us think of many other things besides pyloric obstruction.

It is also interesting to note that one factor may neutralize another; that the rapid emptying of achylia may be slowed down by ptosis or the delay due to obstruction may be compensated by the powerful peristalsis of a strong person.

Chronic Ulcer of the Stomach and Duodenum.—There have been great changes in the last few years in the diagnosis of chronic peptic ulcer. We have recognized the imperfections of some of our older methods of examination, and laid more emphasis on newer ones, such as the *x*-ray. We are now able to locate ulcers, to measure their size, and to study more completely their

effect on the functions of the stomach. We are much better able to classify these ulcers in groups for treatment and to choose the treatment best suited to the individual case. The *x*-ray also helps greatly in diagnosing doubtful gall-bladder and appendix cases which simulate ulcer.

We are now able to follow the ulcers in a new way during their course of treatment, whether medical or surgical, and to see what becomes of them, whether they are getting larger or smaller, not depending wholly on what the patient tells us about his feelings, but *watching the ulcer itself* from time to time and its effect on the functions of the stomach, and changing our treatment to meet the needs of the individual case.

There are great advantages in this method by direct observation of the ulcer and the progress of healing by the Roentgen ray; it helps greatly in deciding for and against surgery. By directly observing the patient we can best protect him from the development of cancer and make an actual, *early* diagnosis of secondary cancer and make a life-saving operation possible.

This is not the place to discuss the technical details of Roentgen-ray examination. I will merely say that in ulcer and cancer the tendency is to base our diagnosis less on so-called indirect signs, such as changes in peristalsis, spasm, and six-hour residue, and lay most stress on the actual demonstration of the lesion itself by finding a defect in the stomach or duodenal wall. The indirect signs are valuable. They serve as a red flag and call our attention to the fact that something is wrong with the stomach which requires explanation, but they appear in *other conditions also* which cause reflex irritation and spasm of the stomach, such as chronic appendix or gall-bladder or a general condition of nervous irritability.

There is no place in the digestive tract where Roentgen signs are so definite as in the duodenum, because nine-tenths of the lesions are found in the first inch or so of the duodenum, and we can concentrate our attention on a very small area; definite evidence of duodenal ulcer is found in 90 per cent. or more of the severe chronic cases.

The chronic indurated ulcer of the stomach along the lesser

curvature or near the pylorus is readily found with the Roentgen ray. *This is our opportunity to prevent cancer* by discovering the *location* and *size* of the ulcer. Size is very important, for more than half the ulcers of the stomach, with a crater as large as a quarter, are becoming malignant. We very rarely discover an early primary cancer, but we can discover and locate the good-sized gastric ulcers and get them to the surgeon before they become cancerous.

The rare ulcers in the middle of the front or back wall of the stomach may be missed on *x*-ray examination because we cannot get them in profile in any position, and we occasionally see a case of hematemesis, with normal *x*-ray pictures, in which the blood "seeps" into the stomach or in which there is a tiny eroded artery or bleeding vein and no true chronic ulcer at all.

It is very important to decide whether delay in emptying is due merely to spasm of the pylorus or to actual tissue narrowing. I want to emphasize here the use of atropin in deciding this question. This therapeutic test is very important—diet, rest, alkalies, and atropin soothe and quiet the irritable stomach, and many a case of obstruction, which we believe at first to be organic. relaxes almost entirely, and is shown to be merely the result of spasm. Dr. W. H. Mayo stated some years ago that delay in emptying the stomach in chronic ulcer is due to actual tissue narrowing in less than 10 per cent. of the cases.

This classification of the kind of obstruction is very important from the point of view of treatment. The cases with spastic delay frequently yield to medical treatment and are suitable medical cases. The cases with organic obstruction rarely yield to medical treatment and usually should go at once to the surgeon. The milder cases yield, the severer ones may not. In short, if a stomach relaxes and empties after the use of atropin we can conclude that the obstruction was due to spasm. If the obstruction does not relax after the use of atropin we have not absolutely ruled out spasm as a cause of delay.

Another important point is that muscular spasm is a very variable, intermittent affair, and a patient with spasm of the pylorus does not always behave alike at different times, entirely

apart from the use of drugs; and in deciding questions about the treatment or operation on such a case more than one examination may be needed.

The Roentgen ray can also be used to great advantage in judging the results of either medical or surgical treatment by detecting the changes which occur in the course of the healing; this includes the changes in anatomy, in peristalsis, and in emptying of the stomach. Peptic ulcers are essentially chronic and should be watched carefully for months to see what becomes of them; an examination with the Roentgen ray should be made several times during the first year after treatment is begun.

The anatomic results of treatment of ulcer are very interesting. They are most easily studied in the ulcers along the lesser curvature which have a definite crater, perhaps some spastic hour-glass deformity, and also in ulcers of the duodenum where there is a characteristic deformity. We have seen the crater of gastric ulcer along the lesser curvature entirely disappear in several cases after medical treatment, but thus far we have not seen a single markedly deformed duodenal cap which has entirely filled out to its former smooth, plump contour after treatment. Evidently a certain amount of scar tissue or adhesions have persisted which still deform the delicate walls of the duodenum. This is true even in cases which have remained entirely without symptoms for a year or more, and which we considered cured. The deformity of the duodenum has greatly lessened, but *not entirely* disappeared. In ulcers at the pylorus without definite crater and with a good deal of induration and scar tissue the anatomic changes after treatment are not striking.

We must give just a word of warning about using the Roentgen ray to study the anatomy of the healing ulcer. We must be very careful and thorough with the Roentgen examination and use the fluoroscope, as well as take plates for record and for detail. We must not be misled by variations in appearance caused merely by different degrees of filling the stomach and duodenum, or different angles at which pictures are taken. It is even possible that food may occasionally stick in the bottom of an ulcer crater and make it appear shallow. Ulcers are

usually less well seen after a gastro-enterostomy, as the barium meal may run rapidly out of the new opening, leaving the stomach only partly filled. These difficulties can be overcome with care. We believe that the Roentgen examination, which has proved so very helpful in diagnosing ulcer, can be equally useful in watching the results of treatment.

Cancer of the Stomach.—The Roentgen ray is our leading method in detecting cancer of the stomach, and is especially useful because the early symptoms and chemical changes due to gastric cancer are often vague; the technical evidence of cancer is absolutely characteristic in only part of the cases; in the majority it must be judged in connection with other clinical data.

In addition to aiding in diagnosis, the Roentgen ray will definitely locate the cancer, show its size and extent, and help decide about operability. Roentgen evidence helps greatly in discovering latent cancer and is an equal help in ruling out cancer in suspicious cases.

Early diagnosis of cancer of the stomach, the great hope of the new method, has not been fully realized. In order to find *early* cancer the patients must be examined more often and earlier than is the rule. The ideal seems to be early examination of every patient of cancer age who has digestive disturbance. Cancer near the pylorus is more likely to bring the patient earlier for examination than cancer elsewhere.

Roentgen evidence of early cancer of the stomach is very scanty. The early anatomic changes, like the symptoms and other signs, are hard to recognize; the earlier the cancer, the less clear the evidence. This is equally true of the small primary induration or the transition stage from chronic ulcer to cancer.

In spite of these difficulties we believe that this method will aid in the detection of early cancer and should be given a trial in every suspected case. If a case remains doubtful after all the evidence is in, clinical as well as radiologic, it should be re-examined at short intervals until a decision is reached.

I am more and more impressed each year with the great difficulties of *early* diagnosis of cancer of the fundus of the stomach or near the cardia. We may have some loss in weight, slight or

intermittent dysphagia, difficulty in passing the stomach-tube, even positive blood tests in the feces, without getting any *definite* evidence whatever by means of the *x*-ray in any position or repeated examination. When we are not suspicious of the fundus, we may overlook cancer even more easily on account of the partial filling and lack of peristalsis and inaccessiblity to palpation.

Roentgen examination helps greatly in deciding about operability by showing the position and extent, the fixation of the growth, and the presence of obstruction or metastases. It may show that a cancer with marked symptoms is small and mobile and situated at the pylorus and ideal for operation. Large or fixed cancers and those in the upper part of the stomach are unfavorable for surgery. Many needless explorations are avoided by *x*-ray examination, but if there is any reasonable doubt, the patient ought to have the benefit of the exploration.

Rarer Conditions.—The *x*-ray examination is particularly valuable in some of the less common or rare conditions of the stomach which are almost impossible to diagnose without it, such as hour-glass stomach, syphilis, foreign bodies, and diaphragmatic hernia.

CLINIC OF DR. W. RICHARD OHLER

BOSTON CITY HOSPITAL

RENAL FUNCTION TESTS—THEIR CLINICAL APPLICATION

The Phthalein Test; the Two-hour Test for Fixation of Specific Gravity; the Blood Urea Nitrogen Determination, and the Study of the Urine for Amount and for Salt and Nitrogen Content. Methods of Making All These Tests, and the Application of These Tests to the Every-day Problems of Diagnosis, Prognosis, and Treatment. Illustrative Cases.

THERE are so many kidney function tests and so much has been written about them that it is, of course, impossible to consider them all in the time allotted. The question that interests the average clinician is the selection of a few such tests, the technic of which is not too complicated, and the application of these tests to the every-day problems of diagnosis, prognosis, and treatment.

The renal function tests which in our opinion are best suited for routine clinical purposes are: (1) The phthalein test, (2) the two-hour test for fixation of specific gravity, (3) the blood urea nitrogen determination, and (4) the study of the urine for amount and for salt and nitrogen content.

The Phthalein Test.—The technic of this test is as follows: (1) Select any two-hour period in the patient's day. (2) Have patient void. (3) Immediately afterward inject exactly 1 c.c. of phenolsulphonephthalein subcutaneously or intramuscularly, preferably in the lumbar region. (4) Immediately after the dye has been given have patient drink two glasses of water. (5) Collect the urine or catheterize two hours and five minutes after dye has been given. In cases where edema is present the dye is

best given intravenously. Extreme care should be taken to inject exactly 1 c.c. of the dye, and to rinse out the bed-pan or urinal into the specimen bottle. (This is of course most important where small amounts of urine are passed.) (6) Make the specimen strongly alkaline, dilute to 1000 c.c., mix thoroughly, and read a sample against a known standard.

A normal individual excretes over 50 per cent. of the dye in two hours. Cases which give readings between 40 and 50 per cent. should be looked upon as border-line cases and their function should be studied by further tests. Cases which give persistently a reading under 40 generally have definite kidney damage. More than one test should always be done. The test gives us information about the kidney at the time the test is performed; the readings will improve with improvement in the kidney condition. Moreover, the phthalein test is influenced by certain extrarenal factors, such as cardiac decompensation; kidney congestion; enlarged prostate; cystitis, especially where there is retention of urine; and often in cases of inanition. In such complications the phthalein test will not infrequently give an idea as to how much of the patient's condition is due to kidney disease and how much to other factors. Finally, in certain types of nephritis the test may be perfectly normal even though severe kidney damage exists. Bearing in mind the above exceptions, it is safe to say that this test is (1) of some diagnostic importance and (2) that it often gives valuable information concerning the progress of any given case. Its prognostic value is doubtful because patients with a very low reading may live for a number of years and because variations in the reading from time to time may be considerable.

The Two-hour Test for Fixation of Specific Gravity.—The plan of this test is to give the patient a moderately high protein diet with known amounts of fluid and if possible known amounts of salt and nitrogen; to allow nothing between meals and nothing from supper on the day of the test until breakfast next day; to make separate collections of urine at two-hour intervals except that the night urine is collected in a single bottle; and to have supper come at least two hours and a half before the last day

collection so that the diet for supper will have no direct bearing upon the night urine. If we take 8 A. M., 12 Noon, and 5 P. M. as the meal hours, then the routine of the day is as follows: Just before breakfact (8 A. M.) the patient voids, and this specimen need not be saved. Following this, urine collections are made at 10, 12, 2, 4, 6, and 8. From 8 P. M. until 8 A. M. the next day the night urine is collected. The last specimen of night urine is passed at 8 A. M. before the patient has breakfast. These specimens of urine are examined for amount, specific gravity, and where possible for salt and nitrogen. The most important part of the two-hour test, however, is the examination of amount and specific gravity.

The characteristics of a normal test are as follows: (1) Water and specific gravity. Prompt renal response to fluid taken in at meals. Day urine amount greater than night amount. Night amount generally not over 400 to 500 c.c. and generally of high specific gravity (1018–1024). Total urine amount generally not less than 75 per cent. of fluid intake. Specific gravity of the different specimens varies with the amount, that is to say, the lower the amount, the higher the gravity. Variations in specific gravity readings of the different specimens should be over eight points, and the more concentrated urine specimens should give a reading of 1018 or over. (2) Salt is eliminated quickly, therefore expect prompt response to meals and also expect considerable variation in salt concentration in each specimen. (3) Nitrogen is eliminated more slowly, therefore expect the highest concentration in the night specimen, and either no retention or very little retention during the twenty-four-hour period.

The two-hour test is of definite value as an aid to diagnosis. In general, it can be said that abnormalities in this test mean kidney impairment. There are, however, certain exceptions. Fixation of specific gravity has been noted in certain anemias and in diabetes. Cases of myocardial insufficiency frequently show fixation of gravity at a high level (1024–1028). Moreover, in cases where renal impairment is shown only in the two-hour test—clinical evidence being absent and all other tests normal—the two-hour test should be done more than once. Finally the

test is influenceod somewhat by the amount of fluid intake and output. Care should be taken to give the patient at least 2000 c.c. on the day of the test. Also cases eliminating much larger amounts of fluid than are taken in (edema) sometimes show a temporary fixation of gravity. The two-hour test is of no great prognostic importance, because fixation of gravity may persist for a number of years before the termination of the disease. The test is of some value as a guide to treatment because with improvement in kidney condition there is often a return, in part at least, of the power to concentrate urine.

Blood Urea Nitrogen.—Normally increased protein intake or increased tissue catabolism results in an increased concentration of nitrogenous products in the blood, and as a result the kidney secretes more. The normal kidney does not maintain an absolute balance, therefore it is often possible to raise the blood nitrogen in normals temporarily. In nephritis very often the kidney cannot excrete increased or even normal amounts of nitrogenous products, hence we have the danger of retention of these products in the blood. Furthermore, in severe nephritis there is often an increased catabolism, and retention of nitrogenous products takes place even though the diet may contain small amounts of nitrogen.

The normal reading for blood urea nitrogen is generally conceded to be between 8 and 15 mgm. per 100 c.c. of blood. Blood readings are of great value as an aid to treatment and prognosis but of only slight value as an aid to diagnosis. This arises from the fact that the blood determination may be normal in the presence of a severe nephritis. For example, in certain cases of chronic nephritis with edema the blood reading may be normal up to within a few hours of the termination of the disease. In other types of nephritis, however, we can often foretell the approach of uremia or we can state definitely that improvement is taking place despite the fact that there may be no noticeable change clinically, and furthermore we can use the blood readings to check up the efficiency of treatment. Generally speaking, those cases whose blood urea nitrogen can be controlled by dietary measures have the most favorable prognosis. In cases where

the blood urea nitrogen continues to go up despite proper dietary measures the prognosis is, of course, grave.

In addition to the above tests there are certain other studies which are essential. The simplest is the study of fluid intake and output. Disturbance in fluid elimination is one of the early evidences of damaged kidneys. As yet we have no way of determining just how much fluid to allow in a given case. It is not unlikely that in many cases more harm is done by telling patients to drink plenty of water than by a proper restriction of fluid intake. In addition to a study of fluid intake and output, salt and nitrogen determinations will often give valuable information. These determinations should be done over a period of days, during which time the dietary intake is known. A single determination means very little. Very often by such a study we can anticipate improvement in a case owing to the fact that the kidney is excreting larger amounts of salt and nitrogen than it is taking in. The converse is also generally true, except that in those cases where there is an increased breakdown of body protein, in addition to an inability to handle the protein in the diet, blood urea nitrogen determinations will give much more accurate information than urinary studies alone.

The determinations of the blood urea nitrogen and the urinary salt and nitrogen is not difficult. We have had numerous instances where physicians have learned these methods within a two-week period and where subsequent results from their own laboratories have checked well with laboratories of known repute.

In our opinion we can by a proper combination of the above tests and studies receive great help in the management of most cases of nephritis. In the following case reports an attempt will be made to show how these tests apply clinically.

In grouping the following cases no attempt will be made to establish any detailed classification of nephritis. Until such time as we possess more accurate methods for the study of renal disease it seems to us futile to attempt to tie up clinical manifestations with pathologic findings. Perhaps the best we can do at the present time is to classify nephritis into: (1) Acute. (2) Chronic, with edema. (3) Chronic, without edema.

ACUTE NEPHRITIS

Case I.—Joseph M. Age twenty-eight.

History of frequent sore throats during past three years. Peritonsillar abscess May, 1919. Admitted to Hospital June 15, 1919. Three days before admission noticed dull headache and moderate lumbar pain. On morning of 15th awoke to find ankles swollen so badly that he could not put on his shoes, and also noted that there was some puffiness about the eyes.

Physical examination unimportant except for the edema. Urinalysis showed: Specific gravity, 1014; albumin, trace; sediment, many red blood-cells, numerous pus-cells, few granular casts. Blood-pressure 145/100.

As a result of rest in bed, low protein (60 grams) and low salt (2.5 grams) diet, with fluids not over 1200 c.c., his condition rapidly improved, and he insisted upon going home at the end of three weeks. Urinalysis at time of discharge showed: Specific gravity, 1018; albumin, slightest possible trace; sediment, no blood, no pus, an occasional granular cast.

Functional studies were as follows:

June 15, 1919: Phthalein, 15 per cent. Blood urea nitrogen, 39 mgm.

June 23, 1919: Phthalein, 30 per cent. Blood urea nitrogen, 21 mgm. Two-hour test showed tendency toward fixation of gravity (1010 to 1016). The night amount of urine was not excessive, and there was no retention of salt or nitrogen.

Functional tests show definite improvement, but there was considerable evidence of kidney damage at the time of discharge. It was impossible to say at this time whether the patient would make a complete recovery or whether he would progress into a condition of chronic renal disease.

Subsequent History.—On April 8, 1920 he re-entered the hospital solely for renal studies. Since June, 1919 he has had no sore throats (tonsillectomy during summer of 1919), but he suffered a moderately severe attack of influenza during January, 1920.

Urinalysis April 9, 1920: Specific gravity 1024; albumin 0; sediment, no blood, pus, or casts. Blood-pressure, 120/90.

Functional Tests.—Phthalein, 50 per cent.; blood urea nitrogen, 14 mgm.; two-hour test showed no fixation of specific gravity (1010 to 1024), night amount of urine 270 c.c., no retention of salt or nitrogen.

In our opinion this patient has made a complete recovery. In this case renal function studies have merely confirmed the clinical findings, together with the findings from an ordinary routine study of the urine. The patient is being watched carefully, but he is under no dietary restrictions.

Case II.—Lawrence B. Age thirty-eight.

Patient has always considered himself perfectly well. Except for Neisser infection twenty years ago, influenza in October, 1918, and occasional sore throat during August, 1919, there has been no history of disease. Admitted to hospital September 4, 1919. On day of admission noted that ankles and feet were badly swollen, and that there was some puffiness about the eyes. one month previous to this he began to be troubled with headaches, but thought it due to eye trouble. Physical examination was unimportant except for the edema. Blood-pressure 138/112. Urinalysis showed specific gravity 1014; albumin, trace; sediment, many red blood-cells, numerous granular casts.

He remained in the hospital two months, at the end of which time his edema had disappeared, and urinalysis showed: Specific gravity 1018; albumin, slightest possible trace; sediment, rare hyaline cast, no pus, no blood. Blood-pressure 120/80. Fundi normal.

Functional Studies

Date.	Phthalein, per cent.	Blood urea nitrogen, mgm.	Two-hour test.
8/ 4/19	20	29	Specific gravity fixed 1005–1010. Night urine 1015 c.c.
8/15/19	25		Total output 2860 c.c. Total intake 1500 c.c.
8/27/19	23	26	
9/ 4/19	25	29	Specific gravity fixed 1010–1012 Night urine, 700 c.c.
9/25/19	20	31	Total output 1347 c.c. Total intake 1500 c.c.
10/ 6/19	20	20.9	

At the time of discharge this patient showed plenty of evidence of kidney damage. His phthalein remained low, his blood urea nitrogen had fallen only slightly, and his two-hour test showed definite fixation of gravity. Attention is here called to the first two-hour test. Note that the fluid output is much greater than the intake. It is quite possible, therefore, that the very low gravity at this time was due in part to excessive fluid output. The second two-hour test probably gives a more accurate idea of the concentration power of the kidney. Because of the results of functional tests, and because of the slowness with which this patient improved clinically, we felt that there was definite evidence of beginning chronicity in the kidney condition, and consequently advised constant supervision, particularly in regard to diet, physical exertion, and personal hygiene.

Subsequent History.—Patient has been under constant supervision up to June 25, 1920. He is at present doing full duty as a Boston fireman and feels perfectly well. His fundi are normal. Blood-pressure 135/85. Frequent urinalyses, during the past two months, show no albumin, no blood, pus, or casts.

Functional Studies.—Phthalein has gradually risen until it is now 50 per cent. Blood urea nitrogen has been under 15 mgm. for two months. The two-hour test shows: Specific gravity 1006–1020. Night amount 625 c.c. with specific gravity of 1016. Total output 1960 c.c. Total intake 2100 c.c. In other words, this patient's functional tests, together with urine examinations, are now perfectly normal except for slight abnormalities in the two-hour test. We cannot say that this patient has made a complete recovery, but he has made marked improvement. Functional studies in this case have given us a much better idea of the progress of the condition than could have been otherwise obtained. Incidentally this patient exemplifies what can be done for such cases by careful supervision in the Out-Patient Department.

Case III.—Elizabeth S. Age eleven years.

Except for a history of measles in early childhood there has been no previous illness up to the present. Patient admitted to

Hospital May 9, 1920. Previous to admission she had a discharging left ear for two weeks; upon admission she had signs of mastoiditis, and was operated upon at once. At the time of admission she had some slight edema of the feet, and on day after operation there was marked puffiness about the eyes, and it was noted that she was passing blood, pus, and casts in the urine. On May 12th she vomited several times, and there was some slight muscular twitching. Blood-pressure 120/80.

Functional Studies.—May 12, 1920: Phthalein 10 per cent. Blood urea nitrogen 113.7 mgm. From May 11th to May 16th the patient eliminated an average of 1200 c.c. of urine daily on an intake of 1000 c.c.; also during these four days she took in about 20 grams of nitrogen ($20 \times 6.25 = 135$ grams of protein) and 5 to 8 grams of salt in the diet, and eliminated 54 grams of nitrogen and about 10 grams of salt in the urine. She was having frequent watery movements, the nitrogen content of which was not determined. Despite the fact that the initial blood urea nitrogen reading was extremely high and that the patient showed some evidences of beginning uremia, we were able to give a favorable immediate prognosis because (1) the patient was eliminating fluids satisfactorily, and (2) she was getting rid of much larger amounts of nitrogen than she was taking in.

Subsequent events justified this prognosis. By May 17th the blood urea nitrogen was 70 mgm.; on May 24th it was 24 mgm.; and by May 31st it had reached 13 mgm. During the the same time the phthalein had risen from 10 to 30 per cent. Also during this period the patient took care of 2000 c.c. of fluid daily; she eliminated nearly three times as much nitrogen in the urine as the diet contained and a slight excess of salt over the intake. At the present time (June 25, 1920) the patient is free from edema, her blood urea nitrogen remains below 15 mgm., and her phthalein has risen to 50 per cent. Urinalysis shows: Albumin, slight trace; sediment, an occasional red blood-cell, and few granular and hyaline casts. Her blood-pressure has dropped to 110/80.

There are other factors, however, to be considered in giving a final prognosis in this case: (1) At present the patient has the

pale, pasty appearance of a chronic nephritic. (2) At no time during the past few weeks has the specific gravity of the urine been over 1012. (3) Her fundi show a definite albuminuric retinitis. (4) Her two-hour test shows definite fixation of specific gravity (1004 to 1009) and the night urine amounts to 547 c.c.

Despite the fact, therefore, that the blood urea nitrogen and the phthalein are normal, there is plenty of evidence pointing toward beginning chronicity in this case. We know that fixation of gravity is not of definite prognostic value. But the presence of an albuminuric retinitis makes the outlook from the point of view of duration of life very grave. This case illustrates very well the value of studies of nitrogen and salt elimination; it also illustrates the fact that the phthalein and blood urea nitrogen may be normal in the presence of severe kidney damage. With any change for the worse in this patient's condition the blood urea nitrogen will undoubtedly increase.

CHRONIC NEPHRITIS WITH EDEMA

Case IV.—Margaret M. Age twenty.

Patient gives a past history of pertussis in childhood. Several months before admission to the hospital patient underwent a rather intensive course of treatment for syphilis. She had six salvarsan treatments and numerous injections of mercury. About two months before admission she noticed that her ankles began to swell. This symptom constantly grew worse until at the time of entrance into the hospital she was in a condition of general anasarca. Urinalysis at this time showed: Small amount (250 to 400 c.c.); specific gravity 1036; albumin, trace; sediment, numerous red blood-cells, many granular, hyaline, and fatty casts. Patient remained in the hospital nearly five months. At time of discharge urinalysis showed: Amount 800 to 1000 c.c.; specific gravity 1016; albumin, trace; sediment, no blood, few hyaline, granular, and fatty casts.

The problem in the treatment of this patient was to free her of the edema. For two months the patient was kept on a diet low in protein and salt, and moderately high in carbohydrates, with a fluid intake not over 1000 c.c. Also during this time active

catharsis was maintained. At the end of this period there was no change in her general condition. The urine output remained low, never reaching 1000 c.c., and the salt output never went above 2.5 grams for twenty-four hours. For one month following this period the patient was put on a diet containing absolutely no salt, high in carbohydrates, and containing less than 500 c.c. of fluid. For a time the patient lost a little weight on this diet, but the net result was not beneficial enough to justify our continuing it. Restrictive measures as far as salt intake was concerned, therefore, proved to be ineffective. In other words, it was not possible to produce an excess salt elimination over intake.

At various times digitalis was given together with theobromin sodium salicylate (diuretin) in small amounts, but with no beneficial results. The small amounts of diuretin, however, apparently did no harm.

Six weeks before discharge the patient had an attack of phlebitis, and for a few days it was thought that she would die. At this time she was given diuretin, 30 grains, and tincture of digitalis, 1 dram, each, three times a day. Also she was allowed food and drink about as desired. (Protein intake probably never went over 70 grams a day.) Response to drug treatment was very striking. Within three days she began to lose weight. At this time digitalis was reduced to 30 minims three times a day. At the end of two weeks she had lost 26 pounds, and when discharged she weighed 48 pounds less than when admitted.

Functional studies in this case were of interest because they were essentially normal except for the two-hour test. Phthalein output varied from 35 to 55 per cent. Blood urea nitrogen never went over 16.5 mgm. The two-hour test, however, did show fixation of specific gravity on several occasions. We have here a type of nephritis in which functional studies were of but little value.

Case V.—Francis J. Age twenty-two.

Patient gives a history of measles and whooping-cough as a child. In July, 1919 he met with an accident resulting in a

compound fracture of the tibia of the right leg. He was sent to the Marine Hospital, Boston, where he remained practically the entire time up until his admission to the Boston City Hospital in March, 1920. While at the Marine Hospital he had several operations on the lower leg apparently because of sepsis. In August, 1919 there appears a note in the patient's record stating that "Patient's kidney condition prevents operation." Kidney trouble apparently became a complication during his stay in the hospital, but on this point we cannot be certain. At the time of his admission to the Boston City Hospital he was in a condition of general anasarca. His urine showed: Amount about 600 c.c.; specific gravity 1028; albumin, large trace; sediment, occasional red blood-cell, few pus-cells, numerous hyaline and fatty casts. His blood-pressure was 118/60. Except for his edema the physical examination was essentially negative.

Patient remained in the hospital until May 25, 1920, during which time various methods were instituted to rid him of the edema. At the time of his discharge, however, his condition was practically unchanged, and his urine examination gave practically the same findings as upon entrance. For nearly two months he was kept on a low salt, low protein diet, with fluids not over 1200 c.c. Restrictive measures, however, failed to give beneficial results. The reason for this lay in the fact that during this time he eliminated an average of only about 2 grams of salt a day. In other words, it was not possible to bring about an elimination of salt in excess of intake. The patient was also given a course of treatment with digitalis and diuretin. For a time it seemed as if diuretin in fairly large doseas (15 grains three times a day) was beneficial, but the diuretic response was only temporary. During the last three weeks of his stay at the hospital he was allowed a fairly high protein diet with only moderate fluid restriction. On this diet there seemed to be some clearing up of the edema and the patient professed to feel much better. As a result of this apparent improvement the patient insisted on going home. At home he continued to eat heartily and seemed to be doing well until he had a rather sudden change for the worse, and died apparently in uremia.

Renal function studies in this case were of interest because here again they gave us very little idea of the severity of the kidney condition. For example, his phthalein output varied between 30 and 45 per cent. His blood urea nitrogen was always under 15 mgm. until shortly before discharge, when it rose to 17 mgm. His two-hour test showed a distinct tendency to fixation of specific gravity at a rather high level—1014 to 1019. It is barely possible that the slight rise in blood urea nitrogen was of prognostic significance. Unfortunately, however, we were unable to obtain further blood readings after the patient left the hospital.

It is not my purpose in this paper to discuss various methods of treatment. In both of the cases given above restrictive measures failed to have any effect upon edema. In one case a high protein diet together with the use of diuretin seemed to give beneficial results. It will be noted, however, in this case that there was a syphilitic background. Cases with edema resulting from antispecific treatment have been known to clear up spontaneously. In the second case diuretin had no beneficial result, and it is quite possible that the high protein diet was the determining factor in the patient's death. We have seen other cases of this type of nephritis where diuretin and drugs of its class and where a high protein diet were distinctly harmful.

CHRONIC NEPHRITIS WITHOUT EDEMA

Case VI.—Robert B. Age twenty-eight.

Patient entered the hospital November 17, 1919, complaining of headache, vomiting, blurring of vision, and increasing weakness. He gives a previous history of frequent attacks of sore throat during the past four or five years; of a severe attack of furunculosis 1911 to 1913, and of frequent headaches and dizzy spells during the past three years. Furthermore, for a number of years he had been troubled with polyuria and nocturia. At the time of admission his urine showed: Specific gravtiy 1010; albumin, very slight trace; sediment, occasional red blood-cells, few hyaline and granular casts. His blood-pressure was 184/130. There was no edema. His fundi showed marked albuminuric

retinitis. Physical examination was otherwise unimportant except for a slightly enlarged heart.

Renal functional studies at the time of admission: Phthalein 5 per cent.; blood urea nitrogen 116 mgm.; two-hour test, marked fixation of specific gravity at 1008, night urine 750 c.c. He was put on a low protein, low salt diet (protein 60 to 70 grams, salt 3 to 4 grams). Despite the fact that during the first week of his stay in the hospital he eliminated nearly as much nitrogen and salt as he took in, his blood urea nitrogen during this time rose from 116 to 170 mgm. However, the patient seemed better clinically, and as a matter of fact the clinical notes for this period state that he is better. From a laboratory point of view, however, he represented a case where the blood urea nitrogen could not be controlled by dietary measures, and because of this a bad prognosis was given. During the next week the patient was put on a still lower protein and salt diet, but nevertheless his blood urea nitrogen rose from 170 to 255 mgm. and two days later he developed uremia and died.

Functional studies in this case were of distinct value from the point of view of prognosis.

Case VII.—Warren F. Age twenty-one.

Patient entered the hospital November 28, 1919, complaining of headache, blurring of vision, nocturia, and frequent cramps in the legs and arms. Physical examination showed a moderate amount of albuminuric retinitis and slight edema of the ankles. Urinalysis at this time showed: Specific gravity 1010; albumin, slight trace; sediment, occasional hyaline and granular casts. His blood-pressure was 164/80.

The diagnosis of nephritis was first made on this case in April, 1917. Between this time and the present he has been a frequent hospital patient, with symptoms and urinary findings very much like the above. A study of the patient's various records illustrates very well both the value and the limitation of renal function tests. For example, from April, 1917 to June, 1920 the phthalein test has been done a large number of times, with readings varying from 5 to 35 per cent. In 1918 there is a

record of a phthalein of 10 per cent., early in 1920 of 5 per cent., and in June, 1920, of 25 per cent. This illustrates the fact that the phthalein is an index of the patient's function at the time the test is done, and that too much emphasis cannot be placed upon its prognostic value. Furthermore, as far back as 1917 the patient probably had a fixation of specific gravity, for in no place do we find record of a reading over 1014. As would be expected, therefore, two-hour tests show definite fixation of gravity and a large amount of night urine. It has already been pointed out that while fixation of gravity may be looked upon as generally showing definite kidney damage, it has limited value as a prognostic sign. The most important functional studies from the point of view of treatment have been the blood urea nitrogen. In December, 1919 he had a reading of 62 mgm., and from this time to February, 1920 the reading rose slowly to 71 mgm. During this time it was difficult to get the patient to stick to his low protein, low salt diet, but a few days after he had been told that his blood urea nitrogen was 71 mgm. he was seized with a convulsive attack. The nature of this attack is not clearly known, neither can we say definitely that it had any connection with the moderately high blood urea nitrogen. Because of the attack, however, the patient became thoroughly frightened, so that he has been very amenable to treatment ever since. As a consequence, since February, 1920 to the present time his blood urea nitrogen has gradually fallen until it is now 31 mgm. and the patient states that he feels much better than he has felt for a number of years. Also during this time his blood-pressure has fallen from 172/120 to 140/100. I believe that this patient represents a type of case where proper supervision in the Out-Patient Department and proper checking of treatment by blood analyses has resulted definitely in prolongation of life.

HYPERTENSION

In a study of nephritis of this sort I believe it advisable to record a few cases of hypertension because of the growing interest in the question of hypertension and its relation to nephritis.

Case VIII.—Ellen D. Age thirty-four.

Patient admitted to the hospital in June, 1919. She gives a past history of measles and scarlet fever in childhood, pneumonia four years ago, typhoid fever eight years ago. At the time of admission she complained only of nervousness. Her blood-pressure was 280/155. Her fundi were normal. The Wassermann test was negative. Physical examination was otherwise unimportant except for a very slightly enlarged heart and an occasional extrasystole. Urinalysis showed: Specific gravity 1011 to 1018; albumin, slightest possible trace; sediment, very rare hyaline cast.

Kidney function studies were as follows: Phthalein 45 to 50 per. cent.; blood urea nitrogen less than 15 mgm.; two-hour test, specific gravity 1005 to 1015, no increased night amount, no retention of salt or nitrogen. Except for a slight impairment of the power to concentrate urine, as shown by the two-hour test, kidney function studies in this case show only the very slightest kidney damage. We are apparently dealing with a case of so-called essential hypertension, and the question arises, Can a diagnosis of nephritis be made, and is it necessary to put the patient on a restricted diet? I believe that the physician is assuming a grave responsibility when he tells such patients that they have no kidney trouble. It is quite true that the end-result in these cases is more likely to be due to a cerebral hemorrhage than to impaired kidneys. However, until we have more data on this type of case and particularly data covering a period of years, I think it advisable to keep such patients on a moderately low protein and salt diet. Incidentally, such a diet over a considerable period of time will often result in lowering the blood-pressure. This patient has refused to consider dietary treatment. She is still able to continue with her work. Her blood-pressure at present is 300/160.

Case IX.—H. M. C. Age sixty-five.

Patient entered the hospital in May, 1920 complaining of failure of vision, headache, dizzy spells, and occasional nocturia. These symptoms have been present for a number of years, but

have only become troublesome during the past year. Physical examination shows a moderately enlarged heart, with an accentuated aortic second, and numerous extrasystoles. His Wassermann test is negative. Blood-pressure is 310/160. The fundi show marked arteriosclerotic changes. Urinalysis shows: Specific gravity 1020; albumin, none to slightest possible trace; sediment, rare hyaline cast.

Kidney function studies on this patient gave the following result: Phthalein 40 to 45 per cent.; blood urea nitrogen 19 mgm.; two-hour test, specific gravity 1009 to 1018, night urine 1575, total output 1818 c.c., intake 2000 c.c., no retention of either salt or nitrogen. It is safe to say that while there are evidences of kidney damage in this particular case, the primary condition is undoubtedly one of arteriosclerosis. A strict dietary régime in such a patient is probably not as essential as in a younger patient.

This patient's condition steadily grew worse during his stay in the hospital, and he died one month following admission as a result of a cerebral hemorrhage.

The pathologic report on this patient's kidneys is as follows: "Kidneys, combined weight 330 grams. Microscopic examination shows marked sclerosis of the larger arteries, with narrowing of the lumina, sclerosis of an occasional glomerulus with atrophy of the tubule connected with it, and relative increase of the connective tissue around the glomerulus and tubule affected. Small columns of sclerosis extending in from the capsule are thus formed occasionally. Anatomic diagnosis: Moderate degree of vascular nephritis (arteriosclerosis); the larger arteries are involved more than the finer branches of the glomeruli."

The cases recorded above are, of course, selected ones, chosen in order to point out the benefits and the limitations of routine tests of kidney function. It is true that not every case of nephritis will check up so well with laboratory tests. However, in a group of over 100 cases observed during the past year and a half we have found that laboratory tests have been of distinct value. One must always consider the laboratory test as but one piece of evidence in the proper study of any given case, just as one

considers the Wassermann test as one piece of evidence in the consideration of syphilis. Moreover, in dealing with hospital cases we have found that there is a certain psychologic advantage resulting from renal function studies. For example, when a patient appreciates that there is a rather distinct relationship between a low blood urea nitrogen determination and his clinical condition, he becomes deeply intersted in his own laboratory tests and is much more responsive to suggestions as to treatment. Finally, in a disease like nephritis, where there is so much to be learned in regard to diagnosis, prognosis, and treatment, the more complete the study of any given case, the better will be our conception of the disease, and in the long run the greater will be our service to the patient.

CLINIC OF DR. M. J. ENGLISH

BOSTON CITY HOSPITAL

(From the Clinic of Dr. William H. Robey, Jr.)

AN ATYPICAL CASE OF PNEUMONIA

A Discussion in Clinical Diagnosis; the Case Being Type III in Origin, the Physical Findings, x-Ray Reports, and Laboratory Findings All Making it an Interesting Case for Discussion.

STAFF INTERNS AND HARVARD MEDICAL STUDENTS: The following case is presented for discussion to show the necessity for impressing upon the interns and students that a correct diagnosis is not always made from physical signs alone, but must be made in conjunction with a carefully taken history, a complete physical examination, and even other methods, such as bacteriologic, pathologic, serologic, chemical, electric, must be employed to aid in arriving at the same.

New methods of investigation and instruments of precision are increasing daily, and these should be utilized to collect data to improve certainty in diagnosis. We should not only secure facts by the above means, but we should be able to employ them for analysis and reasoning, the result of which is the formation of diagnosis.

Nothwithstanding our effort to obtain data by inquiry or physical signs or laboratory findings, we are often unable to arrive at a diagnosis, usually when the premises for our conclusion are wanting. The patient may omit an important symptom or the symptoms may not coincide with our knowledge of the disease, the physical signs may be obscure, or they may suggest the involvement of many organs, or at the time of the examination the objective signs may not have developed fully, consequently a

provisional diagnosis may have to be made until the characteristic physical signs or laboratory findings make the diagnosis certain.

The case we are about to discuss brings into action the above points and demonstrates some of the routine methods necessary in the present-day medical diagnosis.

Mrs. N., age thirty-four, married, born in New York City, entered the Hospital June 8th.

Complaint.—Shortness of breath and pain throughout right chest.

Family History.—Her father died of Bright's disease at thirty-five; her mother died of cancer at sixty-two; one brother died of influenza at thirty-four; one brother living and well; she has no sisters.

There is no history of diabetes, insanity, nor further history of tuberculosis or cancer.

Marital History.—She has been married thirteen years, has 5 children living and well, four miscarriages, 2 children dead: (1) Pertussis and dysentery at two months, (2) Bronchopneumonia at three years.

Past History.—Her general health has always been good; she had measles, pertussis, and diphtheria in childhood and no other infections during that period; six months ago she had influenza, mild in character and short in duration, with no complications.

Her menstrual history is normal; she denies venereal disease; no history of operations or injuries; one year ago she weighed 90 pounds; she thinks she weighs 85 pounds now.

Present Illness.—On May 30th she states that she was taken suddenly ill with a chill, cough, and pain in her right chest, which was increased by deep breathing; she had a slight cough and "ached all over," her fever was slight and she raised but little sputum; on one occasion she coughed up about a teaspoonful of blood.

Physical Examination.—The patient is a thin, white woman, poorly developed and nourished, of fair intelligence, conscious and rational, moderately prostrated; both cheeks are slightly

flushed and there is a small group of fading herpes on the left upper lip.

Head.—Normal.

Eyes.—Pupils react to light and distance, both are equal; there is no nystagmus, strabismus, or ptosis.

Ears.—Both ear drums are normal.

Fig. 54.—Consolidation of right lung simulating a pleural effusion. Type III pneumococcus.

Nose.—No discharge; there is dilatation of both alæ nasæ.

Sinuses.—No tenderness present.

Mouth.—Upper teeth are false and there is slight pyorrhea of the lower gums. The tonsils and pharynx are slightly red; no exudate or membrane is present; the tongue is dry and brownish coated.

Neck.—No stiffness or rigidity; no palpable glands or enlarged thyroid.

Chest.—Both sides symmetric, ribs prominent; there is limited expansion on the right side, with no bulging of the intercostal spaces on this side; Litten's phenomena is absent on the right.

Lungs.—There is marked flatness over the entire right lung from apex to base, especially over the right base, and slight dulness in the left base from the eighth to the tenth ribs, the

Fig. 55.—Same case, right lung resolving, disproving possibility of a right pleural effusion.

remainder of the left lung is hyperresonant; the tactile fremitus over the entire right lung is markedly increased and slightly diminished in the left base; the breath sounds are intensely bronchial over the entire right lung and bronchovesicular at the extreme left base; the whispered voice corresponds to the bronchial breathing, being very marked over the entire right side of the chest; very many crepitant râles are heard over the entire right

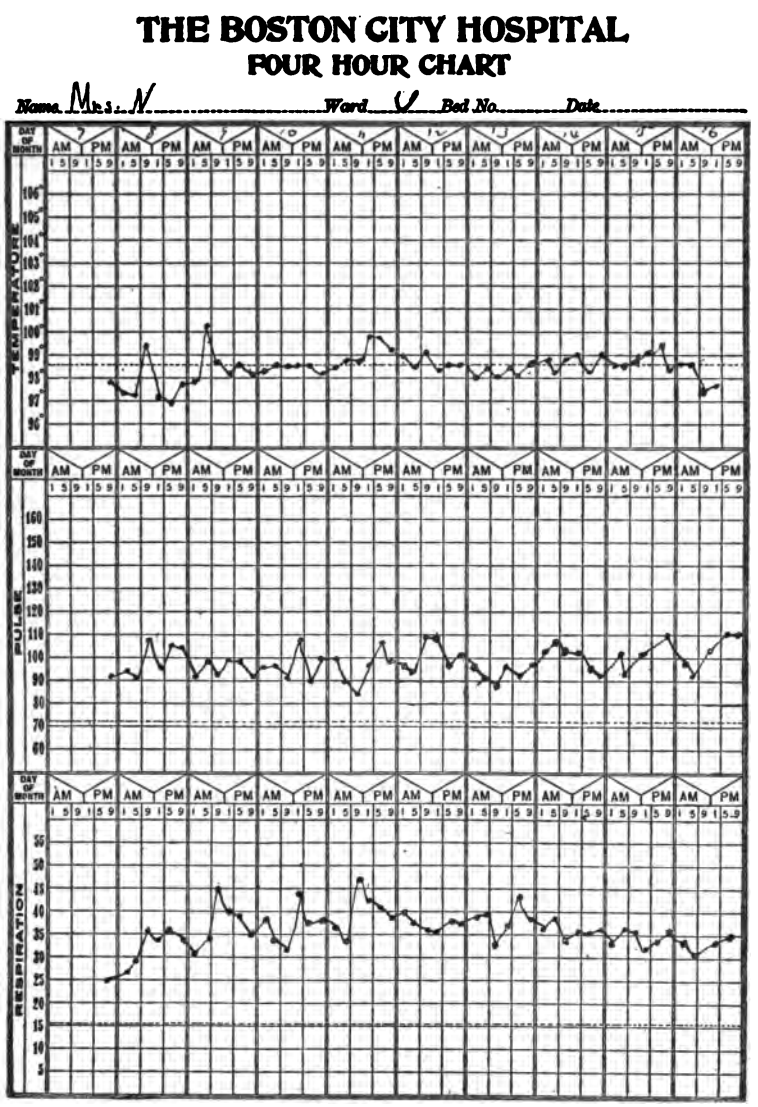

Fig. 56.—Four-hour chart—atypical pneumonia.

lung, being of the consonating type over the right middle lobe and the lower part of the right lower lobe; a pleural friction is heard in the left lower axilla.

Heart.—Apex impulse is barely seen and felt in fifth space 9 cm. from the midsternal line, the right border is not made out,

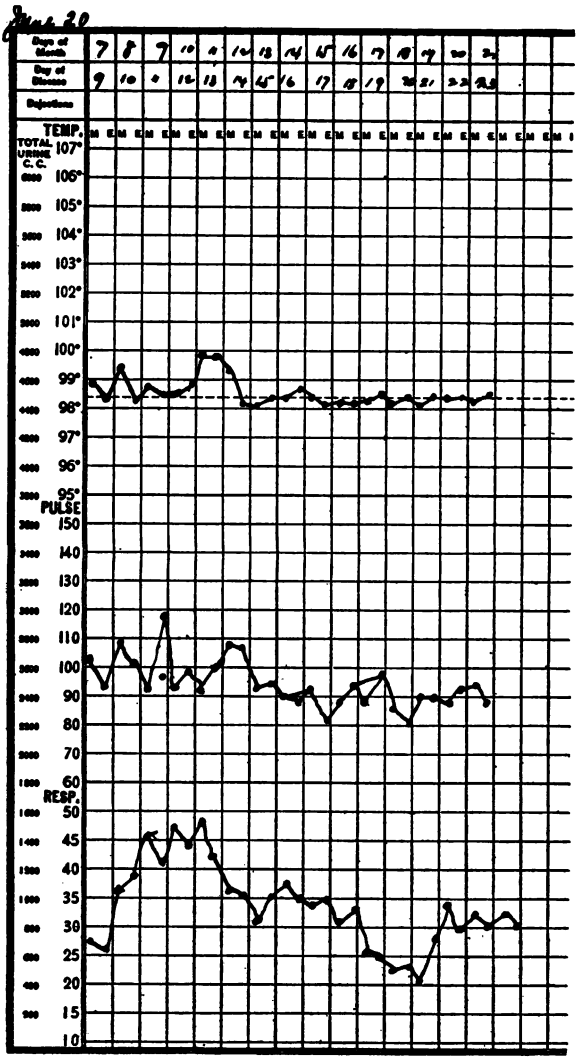

Fig. 57.—Morning and evening chart, same case.

being concomitant with the flatness of the right chest; no thrill is present; the heart sounds are slightly modified, the action is

regular, no murmurs, P^2 is louder than A^2 and accentuated, but not reduplicated. The pulses are synchronous, regular, fair volume, and low tension.

Abdomen.—Soft and tympanitic, no masses, spasm, or tenderness; the liver and spleen are not palpable.

Genitalia.—Normal.

Extremities.—Normal; no edema, reflexes are active.

Skin.—Dry, no eruptions. Slight varicose veins.

The patient's temperature on entrance was 98.8° F., pulse 92, and respirations 26; she was moderately prostrated and dyspneic and slightly cyanosed.

June 9th, day after admission: The urine report was as follows: Slight trace of albumin, specific gravity 1020, sugar absent, the sediment contained many squamous epithelial cells, many red cells, a few leukocytes, an occasional fine and granular cast, and a few renal cells.

The blood-pressure was 110 systolic, 70 diastolic, the white count was 6600, hemoglobin 80 per cent., the Wassermann was negative.

DR. ENGLISH: What are the possibilities for the diagnosis in this case?

HOUSE PHYSICIAN: From the history and the physical examination it seems to me to be a case of acute infection of the respiratory tract, and the possibilities may be confined to a pneumonia due to a pneumococcus or to the influenza or tubercle bacilli, or it may be pleural effusion, serous or purulent, or possibly to tumor of the right lung.

In favor of a pneumococcus or influenzic pneumonia are the sudden onset, with chill, the right chest pain, fever and hemorrhagic sputum; the physical signs are quite typical of pneumonia, the type of which shall have to be decided by a blood-culture, sputum examination, or lung puncture; the herpes labialis, dilatation of the alæ nasæ, the short breathing and cough with tenacious sputum, the flushed cheeks, the rapid respirations very noticeable today but not so marked yesterday. The limitation of motion of the right side of the chest, the flatness of the right chest, with the intense bronchial breathing and loud crepitant râles

over this same area, but more marked at the base, and the increased tactile fremitus and whispered voice sounds make the diagnosis of pneumonia very possible.

DR. ENGLISH: What data would help to confirm it?

HOUSE PHYSICIAN: The finding of one of the four types of pneumococcus or the influenza bacillus in the blood or sputum and the absence of tubercle bacilli in the sputum would help out in arriving at this conclusion.

DR. ENGLISH: What are the possibilities of tuberculosis in this case?

SENIOR INTERN: In favor of tuberculosis, either acute or of long standing, are the preceding history of influenza six months ago which, according to her story, was mild, the duration being only a few days, and the subsequent cough up to the acute onset of the present illness, the hemorrhagic sputum, the low temperature, and the slight febrile progression, with the increased pulse-rate in proportion to the temperature, and the atypical course of the disease would suggest tuberculosis of the lungs. Against tuberculosis would be the sudden onset and the rapid and diffuse involvement of the entire right lung and the negative sputum, though acute tuberculosis cannot be absolutely dismissed from the diagnosis in my opinion.

DR. ENGLISH: Acute tuberculosis usually results from one or more foci in some part of the body or is general in the form of miliary tuberculosis; it may be primary or secondary, and as a rule runs a rapid course. If the lung is involved it is called acute pneumonic phthisis or acute miliary tuberculosis if it involves one or more organs, the latter of which may initiate a fulminating type proving fatal in a few weeks; in the latter, the temperature is usually very high, the pulse extremely rapid and out of proportion to the fever; the prostration, stupor, and emaciation are marked, especially if pericarditis, peritonitis, and meningitis complicate the infection; the physical signs of the pulmonary type of acute miliary tuberculosis are more often those of a bronchopneumonia, namely, dulness with bronchovesicular breathing and crepitant râles following measles or pertussis or some other acute illness; owing to the many forms

in which it may occur it is confounded with other diseases, but a history of a preceding tuberculous infection or the tubercle bacilli in the sputum would determine the diagnosis.

Acute phthisis, popularly known as "galloping consumption," is characterized by a rapid invasion of the lungs, spreading rapidly, the symptoms resembling acute lobar pneumonia, and it is considered as a possible diagnosis when there is no crisis and when there is no evidence of fluid in the chest; the course of the disease is usually downward, though it may occasionally run a slow course, the latter making the diagnosis very difficult. The presence of elastic fibers and tubercle bacilli in the sputum would settle the question.

DR. ENGLISH: Why isn't this a case of pleural effusion?

STUDENT: The acute onset with pain in the right chest, the fever and hemorrhagic sputum, and low white count would suggest an acute pleuritis, later followed by a serous effusion, probably tubercular in origin, because it is the commonest cause of a serous effusion; the increased physical signs and the absence of a displaced heart to the left would be against it; a purulent effusion would follow a pneumonia and would have a high white count; otherwise the reasons against it would be the same as for a serous effusion; the only absolute way to find out is to do a thoracentesis.

DR. ENGLISH: Discuss the possibility of tumor of the lung.

STUDENT: Tumor of the lung is rare, especially at this age; it is usually secondary to involvement of some other organ, and gives rise to cough, pain in the chest, loss of weight, pallor, dyspnea, and cyanosis if there is pressure on structures in the mediastinum; it does not arise with an acute onset, fever, and chill; the physical signs are diminished or absent and very often there is an associated pleural effusion which is hemorrhagic in character similar to a case we now have on the ward; the physical signs in this case are increased and more characteristic of an infection rather than a new growth.

DR. ENGLISH: Tumor of the lung can be eliminated very easily, as the history and signs are those of an infection of acute onset. Tumors of the lung are very often accompanied by a

hemorrhagic effusion which you mentioned, and when this effusion is removed by tapping, the absent signs are still present which is characteristic of this condition from a physical diagnostic point of view.

June 15th. Progress of the case: Five successive reports for tubercle in the sputum have been negative, a second white count is 4200, the urine is normal, the Wassermann negative, and the physical examination of the chest shows that the right lower and middle lobes are slowly resolving, the breathing being now bronchovesicular; the râles are numerous and of the coarse, crepitant type; the tactile fremitus is still marked and the heart is not displaced to the left.

A lung puncture for the bacteriology of the disease was performed today; on entering the pleural cavity no fluid was obtained; 5 c.c. of bloody fluid was removed from the right lung, a smear from which showed a Gram-positive coccus and no tubercle bacilli; the media upon which the fluid was grown was contaminated with Bacillus proteus and so no cultural report could be given.

The sputum today after many trials showed a Type III pneumococcus.

x-Ray of right chest taken the day of admission shows a dense uniform shadow over the entire right lung, with the heart not displaced to the left, the shadow resembling fluid or fluid with a consolidated lung beneath it; a second plate on the 15th of June shows the shadow broken up into smaller but irregular coarse areas, all of uniform consistency, undoubtedly a resolving pneumonia; both plates did not suggest tuberculosis, in fact, an absolute negative diagnosis of tuberculosis was made from both *x*-ray plates.

The patient to date has improved, but the signs in the right lung still show delayed resolution, that is, dulness in the right lower and middle lobes, with many consonating crepitant râles and bronchovesicular breathing, marked consolidation persisting in the right upper lobe; three days after entrance a pleural friction appeared in the left axilla and back with a few râles, but no consolidation, all of which has now disappeared.

The course of the infection since entrance to the hospital is represented in the accompaning charts.

This is an atypical case of pneumonia, Type III in origin; the physical findings, *x*-ray reports, and laboratory findings making it an interesting case for discussion.

I am indebted to Dr. William H. Robey, Jr., for permission to report the discussion of it, and to the house staff for their untiring efforts in working up the case.

CLINIC OF DR. ALBERT A. HORNOR

BOSTON CITY HOSPITAL

ENCEPHALITIS

Full Description of Four Interesting Cases with Full Discussion of Diagnosis, Prognosis, Treatment, and Management.

BEFORE presenting 4 cases of encephalitis which were on the wards of the Fourth Medical Service of the Boston City Hospital at one time this spring it is desirable to call attention to the fact that in the April number of "Archives of Neurology" 85 cases of encephalitis lethargica were reported as having occurred in America since the first report of this disease was made by Bassoe in the Journal of the American Medical Association for April 5, 1919. In addition, Boyd reported in the Canadian Medical Association Journal for February, 1920, 60 cases with 20 deaths occurring in an epidemic at Winnipeg. Furthermore, hardly a European medical journal has appeared in the past year without a report of some cases.

Flexner, in the Journal of the American Medical Association for March 27, 1920, concludes that there is no doubt that encephalitis lethargica is infectious and communicable. His argument that it is a disease entity, and not a sequel of influenza or some other common infectious disease, is certainly convincing. This article by Flexner should be read by all general practitioners, for it is upon them that rests the ultimate responsibility for the prevention of the spread of this disease. He advises that all suspected cases be isolated, and measures be taken to prevent the scattering of nasopharyngeal secretions to other people.

The 4 cases to be described belong rather to the meningoradicular group of Bassoe than to the lethargic type. The cases will be described and then discussed.

Case I.—A married woman, age thirty, was admitted to the hospital March 8, 1920, with the diagnosis of "rheumatism." The chief complaint was pain in her neck. Family and past histories were negative. The present illness began February 27, 1920, with a pain in her right jaw. This was transitory, but followed by pains in all her joints. At the same time she suffered darting pains in her legs, arms, abdomen, and chest, These were particularly marked in the abdominal wall. During the eleven days preceding admission to hospital she lost interest in her home, and, though never stuporous, was quiet. None of her family noted that she was sleeping more than usual.

On admission to the hospital the following abnormalities were found: Temperature 101° F. Pulse 100. Respirations 30. Patient was dull, suggesting typhoid, but could be aroused completely and cried easily. The tongue showed a marked coarse tremor. There was also a coarse tremor of the hands, and if the legs were lifted a coarse twitching of the leg muscles was instituted, which would cease a few moments after leg was allowed to lie free. In addition, coarse jerking movements of the whole body occurred once every three minutes. These movements were all of the same character and did not seem choreiform. The neck was stiff, and attempted flexion caused great pain. The reflexes were normal and there was no ocular ptosis. Examination of the urine showed no abnormalities. Examination of the blood showed a normal number (8000) of white blood-cells. Examination of stained specimen of blood showed it to be normal. Despite this, the stiff neck was considered sufficient indication for a lumbar puncture, which was accordingly done, and a clear fluid under slightly increased pressure was obtained. Examination of this fluid showed no abnormalities. The Wassermann reaction was found to be negative, both blood and spinal fluid. A blood-culture, taken the day after admission to the hospital, remained sterile. Widal reaction was negative on second day in the hospital.

During the first seven days in the hospital temperature remained between 100° and 102° F., more often 101° F., and patient's condition varied little from that found at entrance.

Patient was frequently delirious, but never noisy nor disturbing to other patients in the ward. The case seemed more like one of typhoid fever than any other disease, but there were none of the positive signs of typhoid to be elicited. The white blood count was twice repeated, and was between 6000 and 10,000 both times. No rose spots appeared and the spleen was not palpable. On the ninth day in the hospital patient was extremely dull, pulse-rate 100, and despite the fact that temperature was only 99.4° F., patient seemed quite sick. At this time blood-pressure was 130/80 and the pulse-pressure of 50 led to more hopeful prognosis than might otherwise have been made; furthermore, it was inconsistent with a diagnosis of typhoid fever in a patient as stuporous as was the patient at this time. There were never any abnormal reflexes or changes in sensation. Another sterile blood-culture was obtained on the ninth day in the hospital, and the Widal reaction was again negative on the twelfth day in the hospital.

By the end of the second week in the hospital—the middle of the fourth week of the disease—fever had entirely disappeared. The patient was now quiet and sleeping most of the time, rarely, if ever, changing position voluntarily. She cried easily and felt that she would never get well. The only physical signs present at this time were mask-like facies and marked tremor of tongue. Patient had at no time been hungry, and had to be fed, though once the food was in her mouth she would usually chew a little and then swallow. She would take liquids if given with a spoon. During the next four weeks her interest in herself and surroundings gradually returned. Apparently the loss of strength had not been great, for as soon as she was willing to try to sit up she was able to sit in a chair. Tremor of tongue persisted until the end of the sixth week in the hospital.

She remained in the hospital eight weeks, and on discharge was able to take care of herself. Appetite was good, bowels regular, and she was no longer despondent. After being home a month she was able to do all her housework, and on a visit to hospital, as an out-patient, complained of tiring easily, but otherwise seemed normal.

Case II.—A married man, age twenty-six, a veteran of the recent war, was admitted to the hospital April 6, 1920, with the diagnosis of influenza. On admission he was actively delirious. The family and past histories were negative. The present illness began eleven days before admission with general malaise, hoarseness, and slight fever. A diagnosis of laryngitis was made and he kept at work until four days before admission. At that time he began to suffer pain in right arm and in genitalia. On the same day he became delirious. The activity of the delirium rapidly increased, and for two days before admission he required restraint; nevertheless, during this time he was occasionally rational and completely conscious. The day before admission he was seen by an eminent Boston physician because the family physician thought he might have meningitis. None of the signs of meningitis were found, but there were noted frequent coarse contractions of muscles of trunk and extremities.

On admission to the hospital the following abnormalities were found: Temperature 101.4° F. Pulse 115. Respirations 35. Patient was noisy and actively delirious, requiring constant attention to keep him in bed, but if he answered questions, answers were correct. Frequent contractions of muscles of face, extremities, and of abdominal recti muscles, right more than left, were noted. There was tenderness of muscles (or nerves) of arms and legs; patellar and plantar reflexes were hyperactive. Abdomen was tense and distended—mass palpable below umbilicus, apparently full bladder; 30 ounces of clear urine were obtained by catheter. There was no stiff neck, no Kernig, no abnormal reflexes, and no ocular ptosis. Examination of heart and lungs showed no abnormalities. Repeated attempts to feel spleen were without result.

Examination of urine showed no abnormalities. Examination of blood showed a normal number (7600) of white blood-cells, a negative Widal reaction, and a negative Wassermann. Examination of stained specimen of blood showed it to be normal. A blood-culture done the following day remained sterile.

During the first four days in the hospital delirium became less active and patient was stuporous much of the time. Mus-

cular twitchings became less frequent, save in the recti abdominalis, which became more frequent and regular. Pulse-rate rose to 150; respirations, to 40; and temperature, to 102° F. *x*-Ray of chest made at this time was negative. A lumbar puncture done on the fourth day after admission showed no abnormalities. A blood-culture done the following day remained sterile.

The respiration rate rose to 60 per minute, but no physical signs of trouble in lungs could be found. On the fifth day in the hospital *x*-ray examination of chest was repeated, but showed no evidence of lung involvement. Patient was at no time able to pass urine or feces, requiring twice daily catheterization and frequent enemata. On the seventh day the urine showed a trace of albumin, and much pus and blood. (The bloody urine has been found in cases of encephalitis lethargica not requiring catheterization.) After the sixth day in the hospital it was obvious that patient could not recover. He died on the eighth day in the hospital, or the nineteenth day of disease.

An autopsy showed, in addition to the infiltration of lymphocytes around cerebral vessels, a septicemia of hemolytic streptococcus and a bronchopneumonia of both lower lobes, from which was cultivated hemolytic streptococcus. There was also an acute cystitis.

The hemolytic streptococcus undoubtedly caused the death of this patient, but it does not seem probable that blood-cultures made three and six days before death would have remained sterile if the streptococcus septicemia had been the cause of the original illness. Furthermore, the bronchopneumonia also was probably secondary, for repeated *x*-ray examinations of lungs were negative as late as three days before death.

Case III.—A married man, Russian, age forty-five, occupation a freight handler, was admitted to the hospital April 13, 1920, without a diagnosis. He was admitted to the ward for delirious and noisy patients. Family and past histories were negative. Present illness began ten days before admission, with pains in neck, later pains all over body, especially arms, legs, and head. He developed fever and had to stay in bed. Five days before

admission a low muttering delirium developed, and patient became noisy and unmanageable; for this reason he was brought to the hospital.

On admission to the hospital the provisional diagnosis of edema of the brain, due to alcohol, was made. This was based upon the fact that patient was afebrile, stuporous, and showed no evidence of focal cerebral lesion. The physical examination was otherwise normal. The history given above was shortly obtained, and it was established that he rarely used alcoholic beverages, and then not to excess. A normal spinal fluid was obtained by lumbar puncture. A white blood count showed 8400 cells per cubic millimeter. Stained specimen of blood was normal, Wassermann reactions were negative on both blood and spinal fluids. Examination of urine showed it to be normal. The second day in the hospital it was noted that though patient was often actively delirious, he was at times stuporous, and at that time had coarse twitchings of muscles of face and chest. All reflexes were hyperactive. Urinary bladder was distended, but otherwise physical examination was negative.

This patient became quiet and able to void urine voluntarily during the course of ten days. Then he gradually acquired interest in his surroundings, and at the end of a month was discharged from hospital apparently entirely well.

Case IV.—A schoolboy, age fourteen, was admitted to the hospital April 1, 1920, with a provisional diagnosis of influenza. Family and past histories negative. Present illness began six days before admission, with jerking movements of abdominal muscles and of hands. He developed fever, became restless, and talkative. The coarse jerking movements became more pronounced, and for two days before admission patient was actively delirious and had high fever.

On admission to the hospital the patient's temperature was 101.8° F. Pulse 120. Respirations 34. Physical examination showed patient restless, exhibiting frequent choreiform movements and evidences of irritation of skin due to thrashing around the bed. Patient could not be aroused to complete consciousness

and was at times noisy. Examination of eyes showed no ptosis, no conjunctivitis, and pupils reacted normally. Nose and throat normal. Heart and lungs normal save for increased rate of pulse and respirations. Abdomen was negative save for frequent contractions of abdominal muscles. Extremities showed no tremor, but frequent jerking movements of the adductor muscles of arms and legs. (It was not determined accurately whether all these movements were simultaneous, but this was probably true.) Reflexes were hyperactive. Examination of blood showed: white count 10,800, stained specimen normal, Widal reaction negative, Wassermann negative, and a blood-culture remained sterile. A lumbar puncture was done because patient was febrile and delirious, and no positive diagnosis had been made. The spinal fluid was under increased pressure, but otherwise normal.

The delirium and frequent muscular twitching suggested the diagnosis of chorea major. The fact that the twitchings were bilateral and the same group of muscles always contracted, was strongly against chorea. Other points against chorea major were the height of the fever and the rapidity of pulse and respiration rates. The fact that there was no previous history of chorea was of some, but not great, value.

The initial febrile attack lasted for two weeks after patient came to hospital. During this time acute retention of urine developed, requiring frequent catheterization. The pulse-rate rose to 150 per minute and respirations to 70 per minute and very irregular, but not suggesting Cheyne-Stokes' respiration. There was no cyanosis and both physical and x-ray examinations of chest were repeatedly negative. During the third hospital week this patient apparently improved, only to have a relapse lasting through the fourth week.

For the succeeding six weeks patient had an irregular fever of mild degree due partly, at least, to a complicating cystitis and a bilateral otitis media. During this time he had to be forcibly fed by mouth. He continued to breathe rapidly and slept a great deal. Some days he would awake to full consciousness and be somewhat interested in his surroundings; on other days he

could be aroused only with difficulty, and would promptly return to his sleep. Altogether this patient had fever for two and a half months, and it was not until the end of this time that all muscular twitchings disappeared and he began to improve noticeably.

Now at the end of three months after the onset of the disease he is able to sit up in bed, feed himself, and to void urine and feces voluntarily. He is interested in his surroundings, reads, and watches the hospital interns play tennis. The cystitis and otitis media have subsided. The rapidity of pulse and respiration persists. Respiration rate usually is between 60 and 70 per minute.

The rapid improvement during the time since the fever subsided leads to belief that this boy will eventually get well. How long it will take is uncertain, and if he is able to go to school six months from now his family will be content.

Diagnosis.—The stupor presented in all these cases made the consideration of typhoid fever important. This was ruled out by repeatedly negative blood-cultures and Widal reactions. The diagnosis of meningitis was considered in each case on account of periods of semicoma and periods of restlessness, persistent fever, and the fact that no positive diagnosis had been made. The normal white blood count in every case made the diagnosis of meningococcus meningitis unlikely, but not certainly wrong. The repeatedly clear normal spinal fluid in each case ruled out a meningitis of any sort. The restlessness, delirium, and tremor suggested acute alcoholism, especially in the man with no fever. No history of alcohol helped somewhat in ruling out alcoholism, the age of fourteen practically ruled it out in one case. The fact that when aroused each patient answered questions rationally, and that no hallucinations could be discovered pointed away from acute alcoholism. The diagnosis of chorea major certainly seemed probable at one time in Case IV, but the regularity of the movements, the rapidity of respirations, and the height of the fever were all against this diagnosis. The diagnosis of miliary tuberculosis was suggested in both Case II and Case IV, principally because both patients had a fever of long duration and

no other diagnosis had been established. Inasmuch as this was an entirely negative diagnosis, with a hopeless prognosis, it was discarded and continued study made on the cases. Dementia præcox might well have been considered in Case I had she been seen for the first time during her convalescence. The absence of any demonstrable mental deterioration would have helped to eliminate this disease from the differential diagnosis.

Prognosis.—The mortality of 25 per cent. in this group is about the average of the reported mortalities. Case II and Case IV both seemed dangerously ill from the first. The fact that Case IV is recovering, though his respirations and pulse rate remain high, indicate that involvement of respiratory center has not the same hopeless significance as in infantile paralysis. Both these cases had constant attention from the time of admission to the hospital, both were given large amounts of fluid daily, both were extremely restless, neither could be quieted by ordinary doses of morphin, chloral, paraldehyd, or bromid. Case II had large and frequent doses of morphin and chloral before entering hospital; furthermore he was restrained in bed at home; these procedures surely did patient no good and may have influenced the outcome.

Treatment.—The proper treatment of these cases is of the kind that medical students often consider no treatment, but to the person who has to execute the treatment it seems quite enormous. These patients on account of their delirium need to be watched constantly, on account of their fever need to have their energy conserved by rest in bed, and to be kept quiet. They can easily use up more strength fighting restraint than if allowed to thrash around the bed. Furthermore, if someone is at the bedside to talk with patient he can probably be kept in bed without struggling. In this class of case, just as in typhoid, hydrotherapy seems the best sedative. Woodyatt, of Chicago, by his dehydration fever experiments has emphasized anew the importance of large amounts of fluid in patients with fever. The amount of water, including that in food, taken in health is between 3000 and 5000 c.c. per day. To get this amount of liquid into a patient with fever requires persistent effort on the

part of the nurses or whoever is caring for the patient. Inasmuch as the fever may be of long duration it is desirable to give sufficient calories to prevent great loss in weight. With these principles in mind, and remembering the obligation to prevent the spread of this infectious disease, the following routine is advisable:

1. Isolate patient, and allow no one to leave room where patient is without thoroughly scrubbing hands.

2. Keep patient in bed as long as fever persists, and for at least a week after temperature has reached normal.

3. Have someone in constant attendance as long as fever persists. It is preferable to have trained nurses, but usually, where family alone are available, they can be taught how to do what is needed.

4. Keep a record of fluid intake and keep this at not less than 180 c.c. (6 ounces, one cupful) an hour. If at the end of six hours it is found that amount of liquid taken has been less than 1000 c.c. (1 quart), it is advisable to give a rectal enema of water amounting to about 240 c.c. (8 ounces). This should be repeated in not less than two hours, but should be repeated at this interval often enough to keep fluid intake at 1000 c.c. (1 quart) every six hours. (This will make 4000 c.c. per twenty-four hours.)

5. The liquids taken should contain calories. Cereals, such as oatmeal, rice, cornmeal, Cream of Wheat, may be made into gruels and given in place of water. Cream toast can be made soft enough and given in small enough pieces to be swallowed without chewing. Milk may be given straight or diluted with water. Eggnogs can be given, a dessertspoonful of lactose added to an eggnog will add 50 calories to the eggnog and will not alter the taste. Coffee, tea, and cocoa will serve as vehicles for sugar and cream. Meat broths will not add calories, but will serve to carry down rice. Eggs may be given to the extent of four a day. Whenever patient can and will chew it is advisable to give soft solids, and meat or fish may be given once a day. As soon as temperature has been normal four days an effort should be made to persuade patient to eat solid food. It may be necessary to

put the food into patient's mouth for two weeks after fever has disappeared.

6. Sponge baths with temperature of water (or water and alcohol equal parts) between 90° and 100° F., accompanied by vigorous rubbing, are to be given once in four hours if temperature is 102.5 °F. or above, or if patient is restless.

7. There is no indication for salicylates or other coal-tar products, and hydrotherapy properly employed will obviate the need for opiates.

8. In case of urinary retention try hot packs to lower abdomen for two hours; if this is unsuccessful, resort to use of catheter. The catheterization had best be performed by attending physician, and inasmuch as this can probably not be repeatedly done without infecting bladder, the catheter should be left in place fastened with adhesive plaster. To further help in preventing a cystitis, hexamethylenamin (5 grains four times a day) should be given.

9. To help in the prevention of the spread of encephalitis the following precautions should be observed. All utensils entering room where patient is isolated are to be boiled before being used outside patient's room. Sputum and nasal secretions should be kept covered until burned.

Summary.—Encephalitis has been reported frequently in this country since April, 1919. As Flexner states, it is the duty of clinicians to be on the watch for these cases, and isolate them early. The patient with encephalitis may show no evidence of lethargy when first seen, and excitement may be the more prominent feature throughout the disease. The duration of the disease varies from three weeks to more than two months. The one fatal case in this group died of a complicating septicemia due to Streptococcus hemolyticus. The treatment is similar to that used in typhoid fever, except that patient can take more solid food during height of disease.

CLINIC OF DR. H. ARCHIBALD NISSEN

BOSTON CITY HOSPITAL

CIRRHOSIS OF THE LIVER SHOWING JAUNDICE AND ASCITES. AN ANALYTIC STUDY OF 117 CASES.

Source of Material.—117 postmortem examinations have been made at the Boston City Hospital on cases showing cirrhosis of the liver accompanied with jaundice, ascites, or both. Jaundice was presented by 24 cases, ascites by 37, and 56 cases showed both jaundice and ascites at autopsy. The pathologic records were obtainable for these 117 autopsies, but the clinical records were accessible for only 89. This study was not made with any attempt to solve specific problems; so that an impartial and critical analysis was rendered easier. No datum was accepted that did not seem substantiated by repetition or by clinical, surgical, or laboratory examination.

Classification of Cirrhosis.—Dr. F. B. Mallory's plan of classification of cirrhosis is followed, and his pathologic diagnosis of each liver is the one accepted in this paper.

1. Alcoholic cirrhosis.
2. Syphilitic cirrhosis.
3. Acute yellow atrophy type.
4. Infectious cirrhosis (cholangitis).
5. Hemachromatosis.
6. Primary carcinoma (bile-duct).

The cases of cirrhosis dealt with in this paper are classified under: first, alcoholic cirrhosis 97 cases, clinical records obtained for 77; second, syphilitic cirrhosis (gummata) 10 cases, clinical records for 5; third, cirrhosis (etiology not known but probably healed alcoholic cirrhosis) 11 cases, clinical records obtained for 6. Hemosiderosis was noted 34 times in the 117 cases; it occurred in

26 of the cases of alcoholic cirrhosis and in one of the cases of cirrhosis of unknown etiology. It was present in 13 cases of alcoholic cirrhosis accompanied by jaundice and ascites, in 6 with only jaundice, and in 7 by only ascites. It occurred with ascites in the one case of cirrhosis of unknown etiology.

Alcohol Consumption, Syphilis, Acute Infections.—A history of alcoholic indulgence was obtained from the clinical records in 57 of the 77 cases of alcoholic cirrhosis, in 3 of the 5 cases of syphilitic cirrhosis, and in 5 of the 7 cases of cirrhosis of unknown etiology. There was a positive history of alcohol consumption in 65 of the 89 cases of cirrhosis.

A clinical diagnosis of syphilis was made five times, based on history, clinical examination, and the Wassermann reaction. Most of these cases of cirrhosis were in the hospital prior to the discovery of the Wassermann test, so that it is quite probable that the incidence of syphilis based on the Wassermann reaction alone would be greater had the test been known at that time.

A positive past history of acute infectious arthritis was present in 2 cases, typhoid in 2, erysipelas in 2, and abscess of the neck in 1.

Definition of Alcoholic Cirrhosis.—The actual rôle of alcohol in the production of cirrhosis is not known; but there is a type of cirrhosis of the liver which shows a remarkably constant form of injury to the liver cells, and this type is called alcoholic cirrhosis. Whether alcohol *per se* has any determining influence toward the production of this type of cirrhosis is a mooted question, but, given a history of alcoholic indulgence, time, quality, and quantity consumed do not seem to be of any great determinative value; hence, for purposes of classification the term "alcoholic cirrhosis" is justifiable for cirrhotic livers presenting a similar type of cell degeneration. The amount of cellular change is so varied and the process of such chronicity that many attempts have been made to classify different forms of the same lesion under separate heads. This results in confusion and tends to separate even more pathologist and clinicist. The cellular change consists of the degeneration of cytoplasm into a hyaline meshwork followed by necrosis; later the cell is dissolved by

polymorphonuclear and endothelial leukocytes, and lastly, the hyalin is absorbed, leaving the sclerosis as a final product. The development of the sclerosis in this condition is not fully understood.

This degenerative process may be limited to cells or groups of cells; it may be focal or diffuse; it may show all transitions in the same liver at one time or it may show only the end-result of cirrhosis, which again may be focal or diffuse. The contraction of connective tissue so arranged and distributed in a liver will tend to produce many different shapes. The process is more commonly found around the portal vessels, and obviously the degree of such impairment determines the presence of ascites; and the amount of biliary capillary impairment, the amount of jaundice.

The above makes clear the reason for the confusion in classification of alcoholic cirrhosis; because small livers, large livers, fatty infiltrated livers, with or without hemosiderosis, congestion, smooth or irregular surfaces, focal cirrhosis, general cirrhosis, little or much cirrhosis, are all possible, but each condition is really due to the same predominating characteristic type of cellular injury.

Alcohol (all beverages containing alcohol, including all substances which may be associated with it) as a provocative or direct cause seems to have the most in its favor as an etiologic factor; whether or not a positive history of indulgence is obtained, and until proof to the contrary is shown, it is better to use one general term in description, namely, "alcoholic cirrhosis."

Symptoms and Signs Possible of Production Dependent on an Increased Number of Stimuli Generated in the Liver.—The fact that alcoholic cirrhosis may be of such a protean nature implies the possibility of little or no impairment of physiologic functioning of the liver until the process reaches a stage of development shortly antedating the appearance of jaundice, ascites, or both conditions.

A process that may be of such chronicity and slow development means a gradual adjustment of the body to a new condition. This may be so gradual that the absence of symptoms is to be

expected, or slight changes to be overlooked by the patient, until a sudden increase in the number of stimuli is sent along the nerves of the liver to the cord or brain, producing, in turn, symptoms of disturbance as shown by the presence of tenderness, pain, distress, nausea, vomiting, etc.

The nerve supply of the liver is from, first, the phrenic nerve; second, the parasympathetic fibers of vagal origin, and third, the sympathetic fibers.

The phrenic nerves supply fibers to the capsule of the liver, so that inflammation of the liver or liver capsule, or irritation as from stretching of the capsule, may be sufficient to send afferent stimuli through the phrenic, usually the right, and expressed through the third and fourth cervical sensory nerves; this would mean pain felt over the neck and shoulders in the areas supplied by sensory fibers from the third and fourth cervical cutaneous nerves. The parasympathetic motor and sensory reflexes may be shown by epigastric distress, hyperchlorhydria, nausea, vomiting, constipation. The sympathetic innervation of the liver is from the semilunar ganglion; this, in turn, receives its fibers from the fifth to the ninth thoracic segments of the cord, the same segments supplying the stomach.

Increased stimulation, because of liver changes, could show itself through sensory and motor reflexes, by the spinal nerves arising from corresponding segments of the cord. This would be shown by pain in the right hypochondrium, epigastrium, in the back; in fact, pain may be felt in any area receiving sensory nerves from the fifth to the ninth thoracic nerves, although commonly it is shown in areas supplied by the sixth and seventh thoracic nerves.

Normally we are unaware of any sensation dependent upon stimulation of any of the above-mentioned nerve pathways; for it requires increased stimulation to produce any noticeable reflexes. Acute conditions are accompanied most commonly by sensory and motor reflexes; chronic conditions, by slowly developing organic changes are less apt to produce such reflexes, probably because of the adjustment of the body to the new condition.

Other Factors Influencing Production of Symptoms in Cirrhosis.—The gradual changes in liver function are a progressive chronic process, tending to diminish the normal detoxifying mechanism of the liver. Myocardial, vascular, respiratory changes, congestion in the organs, edema, etc., are usually found postmortem in alcoholic cirrhosis of the liver, and the final picture is one of multiple pathology rather than simply liver pathology.

Frequency of Jaundice, Ascites, and of Both in Alcoholic Cirrhosis.—The 77 cases of alcoholic cirrhosis whose clinical records were obtained showed 44 with both jaundice and ascites at death, 16 with only jaundice, and 17 with ascites alone.

Average Weights of Livers and Spleens in Alcoholic Cirrhosis with Jaundice, or Ascites, or Both Conditions.—Analyzing the above 77 cases, the following was observed:

Both jaundice and ascites:

Liver, 2073 gm.; spleen, 345 gm.

Ascites alone:

Liver, 1960 gm.; spleen, 342 gm.

Jaundice only:

Liver, 2041 gm.; spleen, 327 gm.

This indicates how little difference there is in the weights of alcoholic cirrhotic livers and spleens at the time of death irrespective of the presence of jaundice, ascites, or both conditions.

Time of Appearance of Jaundice and Ascites.—Jaundice and ascites appeared at the same time in 6 cases, ascites appeared before jaundice in 15 cases, and jaundice first in 23 cases.

Symptoms Recorded in the Clinical Records of the 77 Cases of Alcoholic Cirrhosis.—Pain is mentioned thirty-four times, the duration varying from a few days to eight years, constant and intermittent. Pain occurred in 30 cases. This does not include colic-like pain which would be produced by the violent contraction of a hollow viscus, because such pain cannot be originated in the liver. Right hypochondriac pain is mentioned thirteen times; epigastric pain, eight times; general abdominal pain, six times; chest pain, once; lower abdominal pain, twice; small of back, twice; umbilical, once; and the right shoulder, once. Right

hypochondriac, epigastric, and general abdominal pains predominate.

One of the above cases complaining of right hypochondrial pain, which was of two weeks' duration, as well as all other symptoms, showed, postmortem, tuberculosis of the lungs and peritoneum, peritonitis, cirrhosis, and a ruptured esophageal varix which occurred shortly before death.

One of the cases complaining of sudden onset of epigastric pain showed a perforated peptic ulcer and peritonitis, with cirrhosis. It is possible, therefore, that the liver played a minor if any rôle in the production of the pain in the above 2 cases.

Pain in the Right Upper Quadrant.—Twice a right upper quadrant pain appeared as a first symptom, and each time antedated jaundice by five months, ascites not occurring in either case. Twice it appeared one month before ascites, once a few days before jaundice and ascites, once it appeared at about the same time as the ascites, twice a few weeks before jaundice and ascites, three times with jaundice and a few weeks before the appearance of the ascites, once one week before jaundice, and once after it had appeared. Right upper quadrant pain appeared eight times before jaundice or ascites and five times at the same time or after.

Pain in Epigastrium.—It was noted in 1 case four years previous to the appearance of jaundice or ascites as a more or less constant symptom. In 3 cases it was present one year; in 2, one month; and in 2, one or two days before jaundice or ascites (one of these being a perforated peptic ulcer).

General Abdominal Pain.—It appeared either a few days before the onset of jaundice or ascites, or directly after their appearance. Two of these cases showed tuberculous peritonitis.

Chest Pain.—This symptom was due to tuberculosis or pneumonia and was of short duration.

Lower Abdominal Pain.—It was present in 2 cases after considerable ascitic fluid had appeared.

Umbilical Pain.—This condition occurred once with jaundice, and was probably not due to the liver.

It appears that pain is an inconstant symptom and is not

closely related to or dependent upon the size or weight of the liver and spleen. The extent of sclerosis in the liver, rapidity of extension, and accompanying passive congestion probably have a determining part in the development of the pain when present in alcoholic cirrhosis. The above cases direct attention to epigastric pain as possibly occurring some time before jaundice or ascites. Whether active treatment begun at the time of its appearance would have changed the outcome is, of course, debatable; since, even then no doubt, the cirrhosis was too extensive to have affected prognosis greatly.

Observations on Frequency of Symptoms and Signs.—*Vomiting.*—This is noted in 23 cases, duration varying from two days to four years, intermittent or nearly constant in appearance. In 14 cases it appeared previous to the occurrence of jaundice or ascites, and in 9 cases after these conditions had developed.

General weakness is recorded in 16 cases, duration varying from one week to five years, however, with a mean average of months. In 11 cases weakness is complained of before the appearance of jaundice or ascites, in 5 afterward.

Hemochromatosis and Hemosiderosis.—There is no relationship shown in this series of cases between the amount of hemosiderosis in the liver and any symptoms which appear to be dependent for their production on this condition. Only one case showed sufficient deposition of hemosiderin in the skin and liver to be classified as hemochromatosis. The remaining livers showing hemosiderin, but not in the skin, are distributed as follows: 1 case of moderate, 7 cases of slight, and 6 cases of very slight hemochromatosis.

Diarrhea.—This condition is recorded in 15 cases; in 9 cases previous to and in 6 cases after jaundice or ascites appeared. Occasionally it alternated with periods of constipation, although frequently noted with jaundice.

Epigastric Distress.—This was found in 8 cases, in 2 before and 6 after the appearance of jaundice or ascites.

Constipation.—Eight times this was discovered, four times before and four times after the appearance of jaundice and ascites.

Nausea.—This occurred in 7 cases, 4 previous to and 3 after the appearance of jaundice and ascites.

Appearance of Blood from Nose, from Rectum, in Stools and Vomitus.—These conditions occurred twenty-eight times in 20 cases.

Hematesis was noted fifteen times. Fresh blood was raised by mouth or passed through the rectum for the first time in 11 cases, at the time of admission to the hospital or during their stay; 9 of these cases vomited blood, and 2 passed it per rectum. Jaundice alone was present in 2 cases, and ascites alone in 1, both being present in the remaining 8 cases at the time of the passage of blood; 6 cases gave a history of hematemesis with durations and first appearance as follows: once, five years; once, two years; once, a year; and once, six months; once, three months; and once two months previous to the last entry.

All of these cases had both jaundice and ascites at the time of death, average duration fourteen to sixty days. (In each case of hematemesis it antedated the appearance of jaundice or ascites.)

Nasal hemorrhage came four times, each time after appearance of jaundice and ascites.

Rectal hemorrhage and blood in stools was noted ten times singly or in combination. Twice fresh blood from hemorrhage during stay in hospital and eight times changed blood noted after appearance of jaundice and ascites.

Nosebleed.—This was recorded four times—in three before and in one after jaundice and ascites appeared.

Hemorrhoids.—These were present in 9 cases, usually recorded as having been present from one to five years before death.

Distended Superficial Veins.—These were present in 19 cases.

Loss of Weight.—This was noted six times.

Number of Cases Tapped.—26 cases were tapped: 10 only once; 9, twice; 2, three times; 3, five times; 1, seven times; and 1, eleven times. That is, 19 cases were tapped once or twice before death, and 7 three or more times.

Number of Cases Not Tapped.—Ascites was noted in 25 cases that were not tapped. Postmortem 11 more cases were discovered.

Prognosis in Tapped and Untapped Cases.—The total duration of the presence of ascites in the 26 tapped cases was 1196 days and in the 25 untapped cases 822 days.

Weights of Livers and Spleens in the Two Groups.—They were approximately the same.

Jaundice in Tapped and Untapped Cases.—Jaundice was present in 17 of the tapped cases, and not present in 5. It was present in all of the 25 untapped cases.

If the data are studied, several items of interest stand out rather prominently:

First: Hematemesis as a first indication of alcoholic cirrhosis is not borne out by this group.

Second: Jaundice and ascites were both present in 44 cases at time of death.

Third: Jaundice alone was present in 18 cases and ascites alone in 15 cases.

Fourth: After ascites appears prognosis is usually a matter of months, and the majority of cases are not tapped more than twice before time of death. Prognosis in cases tapped and not tapped does not seem to differ greatly.

Fifth: The liver was felt below costal margin definitely in 43 of the 77 cases.

Sixth: The spleen was felt in 6 cases.

Seventh: A correct diagnosis of cirrhosis of liver was made antemortem in 50 of the 77 cases.

Eighth: Only 3 cases had a diagnosis of cirrhosis made previous to last entry in the hospital.

Ninth: Diagnosis of cirrhosis was usually made after jaundice or ascites appeared, and prognosis then is very poor.

Tenth: Peritonitis was not frequent; in the 97 cases it was noted six times at autopsy; of these 6 cases, 2 had tuberculous peritonitis and had been tapped once and twice. Of the remaining 4 cases with peritonitis which had not been tapped, 3 were due to tuberculous peritonitis, and 1 was due to a per-

forated peptic ulcer. Of the 3 untapped cases with tuberculous peritonitis, 2 had ascites and 1 jaundice only.

The average of stay in the hospital is fifteen days for each patient. In this group of 77 cases there were 44 males and 33 females; their ages in decades are as follows:

	Male.	Female.
1st	0	0
2d	0	1
3d	3	3
4th	11	9
5th	13	10
6th	11	9
7th	3	0
8th	0	0

This shows clearly that cirrhosis as an underlying cause of death is most apt to occur in males during the fourth to and including the sixth decade, and in females the same is true.

Cirrhosis of Unknown Etiology.—There were 11 cases in this group, and these probably represent healed cases of alcoholic cirrhosis, that is, complete absorption of hyalin, and a regeneration of liver cells, and later contraction of connective tissue so that the external appearance is similar to that of alcoholic cirrhosis as described. The clinical records were obtained for 6 of these cases.

Sex and Age Distribution.—There were 4 males and 2 females; the ages of the males were thirty-one, forty-two, and sixty-four years; of the females, twenty-three and thirty-one years.

Alcoholic History.—Five gave a positive history of alcoholic consumption of many years' duration.

Clinical Diagnosis.—The diagnosis of cirrhosis was made in 5 cases.

Average Duration of Stay in Hospital.—Twenty-one days.

Pain.—This mentioned as being present in only 1 case, distribution of pain was in the lumbar region, above the crest of the ileum and down the right thigh; this pain had been present nine months. Autopsy revealed cancer of the peritoneum and cirrhosis, the liver probably had little active part in the production of this pain.

Diarrhea.—Present in 1 case of three weeks' duration, eight months before last entry.

Epigastric Distress.—Is noted in 1 case present three weeks, occurring three hours after eating.

Hemorrhoids.—Not noted in any of these cases.

Hematemesis.—Is recorded in 1 case; it occurred twenty-eight days after onset of ascites, during the patient's first day in the hospital. Jaundice appeared one day before death. No signs of varices at autopsy.

Occurrence of Jaundice.—Present in 1 case, noted above.

Occurrence of Ascites.—Present in the 6 cases.

Duration of Ascites.—Question of duration in 1 case, present in the others twenty, twenty-four, thirty-three, thirty-four, and ninety-eight days. One had tuberculous peritonitis, not tapped; the one having cancer of the peritoneum was not tapped, and two others not tapped had streptococcic peritonitis. The three tapped cases show one tapped once, and two tapped twice.

Weights of Livers and Spleens.—The variation in weights of the liver is 1090–1650 grams; spleen, 90–955 grams. The liver was felt twice and the spleen once.

Weakness.—Mentioned in one case, this in a man of eighty years; duration not recorded. It is of interest that this patient had had ascites four years previous to last entry and had not had recurrence sooner.

Hemosiderosis.—Is noted once as marked, or becoming classifiable as hemochromatosis, no jaundice, but with pigmentation of the skin.

Dilated Superficial Abdominal Veins.—Noted once in the case having cancer, edema of leg, and ascites.

One case showed miliary tuberculosis of liver, of uterus, and tuberculous peritonitis; this case showed swelling of leg, duration six months, later extending to belly; no jaundice.

No other symptoms are noted in this group of cases, but the general observations made for the previous 77 cases of alcoholic cirrhosis hold true here generally.

Syphilitic Cirrhosis.—The clinical records were obtained for 5 of 10 cases recorded. The 10 cases showed gummata of liver

in 9, healed gummata in 1, and 1 case is questionable as being syphilitic. Amyloid noted in 2 cases, hemosiderosis in 1; peritonitis in 1, not tapped.

Sex and Age Distribution.—Three males, 2 females; ages of males forty, forty-six, and forty-eight years; females, thirty-four and fifty-three years.

History of Syphilis.—One gave a triple positive Wassermann reaction and one a positive history of syphilitic infection nineteen years before last entry.

Alcoholic History.—Three of the 10 cases gave a positive history of alcoholic indulgence.

Clinical Diagnosis.—A clinical diagnosis of cirrhosis was made in 1 case, and this case had the same diagnosis made three years previous at another hospital, at which time liver and spleen were felt.

Average Duration of Stay in Hospital.—Sixteen days.

Pain.—In one case occurred one year previous to date of last entry, constant, in epigastrium. This occurred in the patient in whom a diagnosis of cirrhosis had been made three years before last entry.

Lower Abdominal Tenderness.—Noted in 1 case; same case mentioned above, present one and a half years before entry to the hospital long before jaundice or ascites appeared.

Weakness.—Noted once, above case, three weeks' duration previous to entry, followed in one week with vomiting, present at time of entry.

Diarrhea and Constipation.—Noted once, then alternated in appearance, first constipation, later diarrhea.

Jaundice.—Noted in 4 cases, no ascites.

Ascites.—Noted in 6 cases, not accompanied with jaundice. In the five clinical records there were 2 cases with jaundice (1 intermittent for years, in the case noted under Hematemesis) and 3 with ascites. The average duration of the ascites was thirty to thirty-five days.

Tappings.—None of the ascitic cases was tapped.

Peritonitis.—Noted in 1 case, with jaundice. This case did not show ascites and was not tapped.

Hematemesis.—Noted in 1 case, that occurred shortly before death, about 2 quarts of blood being vomited. At autopsy this case showed an esophageal varix, ruptured. This is the case having had the diagnosis of cirrhosis made three years prior to last entry; with an occasional tarry stool.

Liver and Spleen Felt.—Liver felt in 4 cases, spleen in 1.

Dilated Veins.—None mentioned.

Hemorrhoids.—Noted in 1 case, with previous diagnosis of cirrhosis.

A study of the above analysis indicates that there are no constant symptoms or signs common, and at present no reliable test of liver function, which is of enough definite value to make an early diagnosis of alcoholic or syphilitic or healed cirrhosis; that is, early enough to be of actual preventive value to the patient. After the appearance of jaundice or ascites, or particularly when both conditions are present, life at best becomes a matter of months' duration. There is, of course, the occasional exception.

Only 3 of the 77 cases of alcoholic cirrhosis had had the diagnosis of cirrhosis made one to two years before their last entry to the hospital. One of the cases of syphilitic cirrhosis had had the diagnosis of cirrhosis made three years previous to last entry. One case had had ascites present for a short time one year before last admission and no recurrence until shortly before last entry.

The chief value of this paper is to direct attention more closely to cases of cirrhosis before jaundice or ascites appear. If the symptoms mentioned are present, a failure to find an organic etiology implies functional disturbance. Functional disturbance so often is overlooked or disregarded as the real onset of disease that its emphasis alone is of sufficient value to justify this analysis of cirrhosis. Nothing is shown more clearly than the hopeless outcome in a case with jaundice and ascites.

I believe that the presence of gastro-intestinal symptoms (often with malaise and weakness), if not remediable by dietetic and hygienic treatment within a few weeks, should be the stimulus for serious consideration of the possibility of cirrhosis.

Treatment should be directed to sparing the liver as much as possible and toward increasing the general tone of the body.

Since we know that cirrhosis is so variable in its extent of liver involvement with an absence of symptoms that clearly imply liver changes until a fairly late stage is reached, just so we have all the more reason to consider cirrhosis more often clinically earlier than is done usually.

Two of the cases of alcoholic cirrhosis who had had omentopexies performed during their stay in the hospital had ascites, were seriously ill, and both died a few days after the operation, although collateral circulation was being established.

Peritonitis is noted in 11 of the 117 cases. Six of these had tuberculous peritonitis; 3 had streptococcic peritonitis; 1 case of syphilitic cirrhosis showed peritonitis; and 1 case of peritoneal cancer showed peritonitis; so that the incidence of peritonitis is not so common in this series, as usually recorded.

Tuberculosis noted elsewhere in the body in 4 cases. This was noted in cases of alcoholic cirrhosis.

Eosophageal plexi were engorged or varices were formed in 16 cases. All of these were in cases of alcoholic cirrhosis except one, which was present in syphilitic cirrhosis.

This series corroborates the view that cases of cirrhosis with ascites are rarely tapped more than twice before death occurs. Peritonitis was not as frequent as others have found it. In those tapped more than twice peritonitis is not recorded once. Chronic passive congestion is noted several times, as well as engorged esophageal plexi; the weights of the livers in these cases varied between 1200–2425 grams and the spleen between 225–630 grams; 4 had jaundice and ascites and 3 had ascites at autopsy.

Symptoms possibly dependent on the liver as a cause are of fairly short duration. The extent of damage to the liver cells is not shown definitely by the size of the liver. However, a palpable liver edge (after ruling out anatomic deformity of body as a cause for the ease of palpation) and accompanied with usually disregarded symptoms should be sufficient to make cirrhosis a diagnosis to be considered. Some of the cirrhoses heal and leave a liver fairly efficient functionally, although the liver edge may be

palpated. It is preferable to make an incorrect diagnosis of cirrhosis and let the patient have the benefits possible because of improved hygiene and diet, than to delay diagnosis until signs are so definitely those of cirrhosis that little can be done for the patient, and in these days that means to direct more attention to preventive medicine, to study symptoms before signs of diseased tissue appear. The patient is then given a real chance to regain health.

CONTRIBUTION BY FRANK B. BERRY, M. D.

(FROM THE PATHOLOGICAL LABORATORY OF BOSTON CITY HOSPITAL)

LOBAR PNEUMONIA

Analysis of 400 Autopsies

Introduction.—During the past twenty-three years 4256 autopsies have been performed at the Boston City Hospital, and in 400 (9.4 per cent.) the diagnosis of lobar pneumonia has been made postmortem as the immediate cause of death. In some years the percentage of necropsies to the total number of deaths has risen as high as 34.4 per cent., whereas during the War it dropped to 2.8 per cent.; the average for the whole period, however, is about 13.5 per cent. The number of deaths in the hospital for this same interval has been about 34,500; hence, had a postmortem examination been made on every case, the condition of lobar pneumonia would have been found in about 3000 cases.

Before proceeding further several vulnerable points in this analysis may be mentioned. First, exactly what does one mean by lobar pneumonia? Should this diagnosis be confined exclusively to cases having a sudden onset and running the classical and well-recognized course? Suppose, as occasionally happens, for example, the patient fulfils all the above requirements and yet at autopsy shows a rapid and fulminating bronchopneumonia. Obviously, then, the classification cannot be made entirely from the clinical side. Conversely, a similar objection holds for a decision based entirely upon anatomic findings, for a bronchopneumonia may be so confluent, homogeneous, and extensive as to involve almost whole lobes and yet otherwise does not in the slightest resemble what one ordinarily thinks of as lobar pneumonia. Like difficulties pertain to bacteriologic differentiation.

Pneumococcus Types III and IV may both produce either a lobar or bronchopneumonia. We think an atypical Type II may do the same, and we do not know as to Types I and II, although we have seen only definite lobar pneumonia caused by them. In addition, occasionally we find what one considers as true lobar pneumonia associated with the streptococcus, staphylococcus, or Bacillus mucosus capsulatus.

Second, much rests in the personal equation of the tabulator as to doubtful cases, vagueness of description, etc. Furthermore, this single equation is multiplied many times when one considers the number of persons concerned in the performance of the autopsies. The Pathological Staff has always consisted of three or four men, with one or two changes each year. To be sure, all have been under a unified control and their work has been carefully supervised, but even so, there are bound to have been variations in the judgment and descriptive powers, as well as in the interest, zeal, and thoroughness of the individuals concerned.

Third, one may say that this is not a fair series, inasmuch as any public institution is bound to receive and care for many persons who are far inferior to the general average; thus, predisposing factors, such as alcohol, exposure, undernourishment, etc., play a more important rôle in our cases than they actually do in the population as a whole. Would not a report based on a large selected group of apparently otherwise healthy people, such as the army, for instance, be more accurate?

Fourth, is this a large enough series to be of any value? It concerns but 400 out of 3000 deaths from lobar pneumonia, about one-eighth. According to the laws of chance, would this be a representative cross-section of the whole?

In reply to these queries all I can say is that I have tried to interpret and tabulate the original protocols as justly and correctly as possible. Granted, many times the descriptions have seemed incomplete or inadequate in some details, but, on the other hand, these necropsies have all been carefully performed by capable men, who, in turn, were always under the direct supervision of others more experienced than they.

As to the last two objections, the first answers itself. The very fact that this series consists of patients of all classes and conditions makes it far more representative and reliable than one based on any chosen group. Similarly, it is just as fair to attempt to draw some conclusions from these quite unselected 400 cases, notwithstanding that they are but a fraction of the whole, as it would be to analyze a group from other institutions even though 100 per cent. postmortem examinations were made.

Sex.—Of our autopsies, 286 (71.5 per cent.) were upon males and 114 (28.5 per cent.) on females. These figures approximate very closely those of Fabyan (75 per cent. males and 25 per cent. females), and from a clinical standpoint, those of Sears and Larrabee, and Chatard, who found 76.3 per cent. and 81 per cent. males respectively. It would therefore appear reasonably safe to assume that lobar pneumonia is about three times as prevalent in males as it is in females.

Age.—The age was stated in 387 of the cases:

Age.	Cases.	Per cent.
0– 5	14	3.6
5–10	6	1.6
10–20	8	2.0
20–30	40	10.3
30–40	79	20.4
40–50	83	21.4
50–60	77	19.9
60–70	55	14.2
70–80	21	5.4
80+	4	1.0

Clinically, lobar pneumonia is relatively common in children under the age of six. After that it diminishes up to the age of fifteen, and then gradually increases until early middle life, when it begins to decrease again. This would account in part for our greater number of necropsies on children under the age of five than between the ages of five and ten. But another factor also enters to a slight degree. Included in this series are autopsies both from the main hospital and from the contagious department of about 300 beds. Thus a few of our cases are drawn from what in some instances appeared anatomically to be a true lobar

pneumonia as a late sequel to diphtheria, scarlet fever, measles, or pertussis.

As for the balance of the table, it agrees surprisingly well with what clinical statistics tell us: lobar pneumonia is a disease of young adults and diminishes in its occurrence after about the age of forty-five. Its mortality, however, steadily rises. This decrease in incidence may be ascribed to several causes. First, with the approach of middle life the chronic, metabolic, and neoplastic diseases increase rapidly. Second, with each succeeding decade there are fewer people alive. Third, with advancing age exposure to infection diminishes. And, finally, there appears to be an augmented resistance to lobar pneumonia after middle life.

To illustrate this, we find by referring to the American Experience Mortality Table that out of 100,000 people alive at the age of ten there will be but 92,637 living at the age of twenty and 85,441 at the age of thirty—an average of 89,039 for the decade. During these ten years the incidence of pneumonia is very near its zenith. Let us assume, then, that in this period 220 cases occur. This is the average for this interval of Sears and Larrabee's and Chatard's groups of 1607 cases. Now between the ages of fifty and sixty there are but 63,861 people living; hence if the incidence of the disease remained constant, we should have about two-thirds as many cases, or 173. Instead, using our same standard, we have but 68—slightly less than one-third. To go still further, during the decade from sixty to seventy there are 48,243 people living. Again, were our ratio constant, we should expect a little more than half the number of cases we had between twenty and thirty, or about 115. As a matter of fact, we find 50, or less than a quarter. From this it is evident that other diseases alone cannot account for this wide discrepancy, and so our other two factors of lessened exposure and increased immunity must be considered.

Lung Involvement.—There was a unilateral lung involvement 321 times, and a bilateral 79. The process was confined to the left side in 111 instances and to the right in 210.

Left lung.	
Upper lobe	18
Lower lobe	39
Both	54
Total	111

Right lung.	
Upper lobe	55
Middle lobe	7
Lower lobe	32
Upper and middle lobes	14
Upper and lower lobes	12
Middle and lower lobes	17
All lobes	73
Total	210

Bilateral or Double Pneumonia.—Of the 79 cases of bilateral or double pneumonia, the following combinations were noted:

	Ours.	Fabyan's.
Left upper and right upper	4	3
Left upper and right middle	0	1
Left upper and right lower	4	1
Left upper and lower with right upper	5	2
Left upper and lower with right middle	0	1
Left upper and lower with right lower	5	6
Left upper and lower with right upper and middle	2	2
Left upper and lower with right upper and lower	2	5
Left upper and lower with right middle and lower	4	1
Left upper with right upper and middle	1	2
Left upper with right upper and lower	1	1
Left upper with right middle and lower	1	0
Left upper with all right lung	1	6
Left lower with right upper	7	7
Left lower with right middle	0	1
Left lower with right lower	8	3
Left lower with right upper and middle	4	1
Left lower with right upper and lower	6	3
Left lower with right middle and lower	7	3
Left lower with all right lung	9	12
All lobes both lungs	8	7
Totals	79	68

From a combination of all the above the order of frequency for each lobe affected is as follows:

Right upper	204
Right lower	190
Left lower	160
Right middle	148
Left upper	110

Again, as to the number of lobes involved:

One lobe	151
Two lobes	120
Three lobes	103
Four lobes	18
Five lobes	8
Total	400

Several interesting inferences may be drawn from the above. First, lobar pneumonia tends to be unilateral and to involve but one or two lobes. In over a third of our cases only one lobe was affected, and in almost two-thirds the process did not include more than two lobes. Some maintain that if a careful search is made small patches of consolidation may be found in each lobe in almost every case of lobar pneumonia. The only way to prove or disprove this claim would be to examine histologically innumerable sections taken from innumerable levels and areas in both lungs. As far as the gross picture is concerned, however, we feel that our conclusion is fairly accurate.

As to actual lung and lobe involvement, it is seen that the right lung is much more frequently affected than the left, in a ratio of about 2 to 1. This coincides with the findings both of Sears and Larrabee, and of Chatard. Clinically it appears that the right lower lobe far outdistances the right upper in frequency of infection, whereas from a postmortem standpoint the upper is not only on a parity with but well ahead of the lower. One cannot help feeling from this that when the upper lobe is concerned the prognosis is somewhat more serious than it is when the lower or middle lobes are affected.

In bilateral or double pneumonia the more usual combinations met with would seem to be those involving all five lobes; the entire right lung and left lower lobe; the left lower and right upper or lower lobes; the left lower and two lobes on the right; and the entire left lung with the right upper or lower lobes.

Gross Pathology.—The macroscopic picture varied from the stage of red hepatization to that of organization.

Red	212
Red-gray	225
Gray	317
Organizing	2
Combinations of above	44
Abscess or gangrene	19
Infarction	6

The unaffected lung tissue was described as congested or edematous 167 times. Through errors of omission, however, this figure is probably much smaller than it should be. Bronchopneumonia in another lobe was found in 62 instances (15.5 per cent.).

Other accompanying pulmonary conditions were mentioned, though probably less often than they actually occurred:

Bronchitis	179
Enlarged bronchial glands	86
Tuberculosis (healed and active)	79
Emphysema	22

The Pleura.—The different types of pleural exudate encountered present a rather confusing picture, due largely to the often complicating factor of an old fibrous pleuritis. The left pleura was affected 48 times, the right 97, and there was bilateral involvement in 228 instances.

	Unilateral.	Bilateral.
Fibrin	121	40
Fibrous	126	71
Fibrin and fibrous	65	9
Serofibrin	18	2
Hydrothorax	5	3
Hemothorax	2	0
Totals	337	125
Empyema (total, 59—14.7 per cent.)	54	5
Totals	391	130

Earlier in this paper I have stated that the pneumonic process was confined to one side of the chest in 321 of the bodies examined and was present in both sides in the remaining 79; thus the total

number of lungs in which this condition was found is 479. By a similar computation it is seen that the pleura showed an acute reaction of one sort or another in 383 instances (67.6 per cent.), an old fibrous condition in 268, and was apparently normal in the other 149.

Accompanying conditions and complications in other organs directly associated with the pneumonia and without other ascribable cause compose a rather formidable list:

Jaundice	26
Acute myositis (rectus abdominis)	6
Acute arthritis	1
Superficial abscesses	3
Subdiaphragmatic abscess	1
Retroperitoneal abscess	1
Acute mediastinitis	11
Acute peritonitis	16
Hydropericardium	61
Petechiæ of pericardium	8
Pericarditis, serofibrinous	21
Pericarditis, purulent	21
Cardiac hypertrophy	98
Cardiac dilatation	6
Toxic change of myocardium	15
Abscess of myocardium	1
Thrombi in heart	3
Acute endocarditis	14
Enlarged spleen	192
Toxic change of liver	189
Toxic change of gastro-intestinal tract	47
Toxic change of pancreas	26
Toxic change of kidneys	157
Acute nephritis	4
Abscess of kidneys	3
Adrenals enlarged or congested	3
Acute prostatitis	4
Ovarian abscess	1
Meningitis (277 heads examined)	14
Brain, hemorrhages	4
Brain, abscess	1
Acute sinusitis	2
Acute otitis media	27
Acute mastoiditis	9
Acute parotitis	2

All of the above changes are gross conditions. Only the kidneys were verified microscopically. Parker and Graham, however, have pointed out that the pancreas and adrenals both commonly exhibit toxic lesions with pneumonia. Two other observations are worth noting. First, large, edematous, or intensely congested kidneys do not mean acute nephritis. Not infrequently in lobar pneumonia the kidneys weigh from 400 to 600 grams from edema or congestion alone, and not even histologically can anything further be found. Second, the low percentage of accessory sinus and middle-ear infection is surprising, especially when compared with cases of influenza-pneumonia, where, out of 200 autopsies, acute sinusitis—usually multiple—or otitis media was found in 60 per cent.

Concurrent Diseases.—A large variety of concurrent diseases were found, but all quite independent of the existing pneumonic condition:

Adhesive pericarditis	8
Tuberculosis, spleen	1
Tuberculosis, intestine	4
Amyloid spleen	3
Amyloid liver	1
Amyloid kidneys	2
Liver, cirrhosis	7
Liver, gumma	1
Liver, cyst	1
Intestine, ulcers	4
Pyelitis	2
Arteriosclerosis	134
Aneurysm	3
Cerebral softening	2
Septic endometritis	1
Pus tubes	2
Cholelithiasis	1
Typhoid	2
Purpura	1
Erysipelas	1
Diphtheria	2
Neoplasms	16

Summary and Conclusions.—Do all these figures and tables mean anything? Can any reliance be placed on them or any

conclusions drawn? To a moderate degree, yes. This series of 400 necropsies is sufficiently large and is a fair and representative collection. The few outstanding features may be briefly summarized.

1. Lobar pneumonia is far more prevalent in males than in females, approximately in the ratio of 3 to 1.

2. It is a disease of early and early middle adult life. Its greatest incidence is between the ages of twenty-five and forty-five. After that it gradually diminishes, due partly to the growing inroads of other diseases, and partly to a decrease in exposure and an increased resistance, both of which develop with advancing age.

3. The right lung is affected more often than the left, and the disease tends strongly to be unilateral.

4. Lobar pneumonia, unlike influenza-pneumonia, is distinctly unilobar in type. In about 67 per cent. of cases, both clinically and at autopsy, not more than two lobes have been involved.

5. The pleura shows a reaction so frequently that it may well be considered as an almost constant accompaniment of the original process.

6. The pericardium is affected in varying degree in almost a third of the cases.

7. The products formed or eliminated by or as a result of the pneumococcus, and occasionally other organisms, in lobar pneumonia produce a general toxemia with definite reactions and lesions in many organs of the body. Grossly these changes are best and most frequently seen in the spleen, kidneys, liver, and heart, whereas histologically they are found still more commonly in the adrenals and pancreas.

8. True acute nephritis as a complication or sequel of lobar pneumonia is unusual if not rare.

9. Acute sinusitis and otitis media are decidedly uncommon in lobar pneumonia in adults. This would strengthen the idea suggested by Blake and Cecil that the pneumococcus by itself is of relatively low pathogenicity for the upper respiratory tract.

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FROM THE MEDICAL CLINIC OF THE CHILDREN'S HOSPITAL

Constipation and Eczema in an Infant from an Excess of Fat in Modified Milk

By John Lovett Morse, M. D.

1. Congenital Atelectasis. 2. Bronchial Tetany

By Lewis Webb Hill, M. D.

Two Cases of Acquired Heart Disease in Childhood

By Edwin T. Wyman, M. D.

Chronic Intestinal Indigestion from Starch, Showing Indican Reaction. Report of 33 Cases.

By Karlton G. Percy, M. D.

Enuresis

By Joseph I. Grover, M. D.

A Case for Diagnosis

By Philip H. Sylvester, M. D.

CLINIC OF DR. JOHN LOVETT MORSE

CHILDREN'S HOSPITAL

CONSTIPATION AND ECZEMA IN AN INFANT FROM AN EXCESS OF FAT IN MODIFIED MILK

THIS little girl is three and a half months or, more accurately, fifteen weeks old. She is the fourth child of healthy parents. They are all alive and well and there have been no miscarriages. She was born at full term after a normal labor, was normal at birth, and weighed 7 pounds. She is brought here because of an eruption on the skin, constipation, and failure to gain satisfactorily.

On looking her over we find that she is small, but well nourished. Her color is good. The skin on the cheeks, buttocks, thighs, and elbows is a little reddened and thickened. The anterior fontanel is level. The mouth is healthy. There is a slight rosary. The heart and lungs are normal. The level of the abdomen is that of the thorax; nothing abnormal can be detected in it. The lower border of the liver is palpable 2 cm. below the costal border in the nipple line. The spleen is not palpable. The extremities are normal. There is no spasm or paralysis. There is no enlargement of the peripheral lymph-nodes. She weighs 10 pounds.

It is evident from this examination that she is not a seriously ill baby. She is somewhat underweight, but, in general, her nutrition and development are satisfactory. She has, of course, a slight, rather widely distributed eczema. The rosary shows that she has rickets, but, as this is slight and there are no other bony signs of rickets and no marked disturbance of nutrition, we

are justified in concluding that the rickets is of no practical importance. There being nothing in the physical examination to account for her failure to gain more rapidly, it is almost certain that the cause is to be found in her feeding. The eczema is additional evidence in this direction, because in many instances in infancy eczema is due to some error in the composition of the food. This error is quite frequently an excess of fat. The story of constipation also suggests that the fat in the food may have been excessive, as an excess of fat is one of the common causes of constipation in infancy. Unfortunarely, her mother did not bring one of her stools and she has not had one since she came to the hospital. We cannot, therefore, find out now whether or not our suspicions are correct. If an excess of fat in the food is the cause of her troubles, we can explain her failure to gain properly by saying that the undue richness of the food took away her appetite and prevented her from taking a sufficient quantity. We can also explain the rickets by saying that the excessive amount of fat in the food, by combining with calcium to form soap stools, prevented the absorption of a sufficient amount of calcium to form the bones properly. These suppositions are all theoretic, however, and further study of her feeding history may show that they are unjustified.

It is of little value in a case of this sort to study only the last food. To arrive at satisfactory conclusions it is necessary to study in detail the feeding history from the beginning, although to do this may take considerable time. The results usually justify the trouble, however.

She was breast fed entirely for five weeks. She lost 8 ounces the first week and then gained ½ pound a week. It is said that there was plenty of breast milk, but that it was of poor quality. On questioning the mother more closely, however, we find that there was no real basis for this statement, except, perhaps, that the baby was not gaining rapidly. It was not weighed regularly before or after each nursing to determine just how much it was getting, and the milk was not analyzed. A modified milk mixture was then given in place of one nursing, then in place of another and another, until she was finally weaned entirely when seven

weeks old. During these two weeks she began to be constipated, but continued to gain ½ pound a week. It was, as I am sure you all know, a great mistake to wean this baby without any attempt to determine the quantity and quality of the breast milk or to correct its defects, if there were any. This ought to have been done, because the facts that she did not vomit and had normal stools show that the milk agreed with the baby and evidently did not contain an excess of any element. If the quantity was deficient, it would have been easy to give the baby some modified milk after each feeding, and if the milk was deficient in any element, it would have been easy to give a higher percentage of this element in the artificial food. It is very probable, moreover, that proper attention to the mother's diet and mode of life would have improved the quantity and quality of her milk so that she could have nursed the baby satisfactorily without the addition of an artificial food.

The mixture which she was given when she was weaned was:

Top 8 ounces from 2 quarts	14 ounces
Lime-water	2 "
Water	24 "
Milk-sugar	4 level tablespoonfuls

Let us see what this food really was. The top 8 ounces of a quart of average milk contain 13.3 per cent. of fat. As the milk-sugar goes into solution, the total quantity of the mixture is 40 ounces. The fat content of the mixture is, therefore, 14/40 of 13.3 per cent. of fat, or 4.65 per cent. This percentage of fat is, of course, too high to give any baby and far too high to give to a baby of seven weeks. The top 8 ounces of a quart of milk contain approximately 4.50 per cent. of milk-sugar. The 14 ounces of top milk puts, therefore, 14/40 of 4.50 per cent. of sugar, or 1.50 per cent. of sugar, in the mixture. There are between 3 and 4 level tablespoonfuls of milk-sugar in 1 ounce. A little more than 1 ounce of milk-sugar was thus added to the mixture. One ounce of sugar in a 40-ounce mixture gives 1/40 of 100 per cent., or 2.50 per cent. The percentage of sugar in the mixture is, therefore, 4. The top 8 ounces of a quart of

milk contain a little less than 3.20 per cent. of protein; 14 ounces of this top milk in a 40-ounce mixture puts 14/40 of 3.20 per cent. of protein in the mixture, or 1.12 per cent., that is, for practical purposes, a little more than 1 per cent. On general principles 4 per cent. of milk-sugar is rather low, and 1 per cent. of protein is about right for a baby of this age. The 2 ounces of lime-water in the mixture is of little importance, because, as you know, unless the amount of lime-water is at least 25 per cent. of the amount of milk and cream in the mixture, it has very little effect in delaying the action of rennin on the casein. In this mixture the lime-water is only 14 per cent. of the top milk.

The baby was at first given ten feedings of 3 ounces at two-hour intervals, or a total of 30 ounces in twenty-four hours. This seems like a large amount of food for a baby of this age when its high fat content is considered. It may not be too much, however, as the percentage of sugar is so low. It is, as you know, not the quantity of food which nourishes a baby, but its content in calories. In order to determine whether the baby is getting enough or too much nourishment in this amount of food, we must find out its caloric content. Thirty ounces equal 900 c.c. As the food contains 4.65 per cent. of fat, there are 4.65 grams of fat in each 100 c.c. of food, or 41.8 grams of fat in 900 c.c. of food. The value of 1 gram of fat being 9.3 calories, the fat in the mixture furnishes 388 calories. Figuring in the same way, the mixture contains 36 grams of milk-sugar and 10 grams of protein. The caloric value of 1 gram of sugar and of protein is 4.1. The sugar and protein add therefore (36+10) x 4.1 calories, or 188 calories, to the value of the mixture. The total value of the food is the sum of the fat, sugar, and protein, or 576 calories. The baby weighed 7 pounds at birth, lost ½ pound the first week, and gained ¼ pound a week for six weeks, thus weighing when she began to take this food 8 pounds. She thus got somewhere in the neighborhood of 72 calories per pound when she began to take this food. A kilogram being 2.2 pounds, 8 pounds is equal to 3.6 kilograms. This food gave her, therefore, 160 calories per kilo. This is more than the average baby of that age usually takes, but it is possible that she might utilize it, if she was un-

usually active and might need it to make up the weight which she had not gained.

She took all of this mixture eagerly and gained rapidly for a couple of weeks, so that she was then given eight feedings of 4 ounces at two and one-half-hour intervals, or 32 ounces in twenty-four hours. This amount probably gave her about the same number of calories per kilogram of weight, as she had gained weight in the meantime. She took the extra amount very well for about a week and continued to gain. Then her appetite began to drop off a little and she ceased to gain. She did not vomit, but became very constipated. Her bowels did not move without the aid of suppositories or enemas of oil or glycerin. The mother is not very clear as regards the character of the stools, but thinks that they were smooth and rather light in color. A rash also appeared on her face, which varied from day to day, being sometimes better and sometimes worse. The loss of appetite was probably due in part to the high percentage of fat in the mixture and in part to the excessive caloric value of the food, the total being more than she could be expected to take continuously. The constipation and the rash on the face are readily explained by the high percentage of fat.

When twelve weeks old she weighed 9½ pounds. The amount of top milk was then increased by 2 ounces and the same amount of water was left out. The sugar was also increased. The mixture was then

Top 8 ounces from 2 quarts.....	16 ounces
Lime-water.....	2 "
Water.....	22 "
Milk-sugar.....	3 rounded tablespoonfuls

She was given eight feedings of 3¾ ounces at three-hour intervals, being awakened at night to be fed. This gives a total of 30 ounces in twenty-four hours. It is rather difficult to see why this change was made, unless it was that the physician thought the baby would gain more if the food was stronger, and that her appetite would be better if the intervals were longer and the amount given at a feeding smaller. It is evident that he did not

appreciate the true situation at all and did not understand what changes, if any, he really made in the composition and caloric value of the food. Let us see what this new mixture really was. Figuring it in the same way as before and calculating that a rounded tablespoonful of milk-sugar weighs ½ ounce, we find that the composition of the mixture is:

	Per cent.
Fat	5.32
Milk-sugar	5.55
Protein	1.28

The lime-water is 12.5 per cent. of the top milk in the mixture and the caloric value of the mixture is 725 calories. If she took the whole mixture she would get 168 calories per kilogram of body weight. The net result of these changes was, therefore, to exaggerate all the bad points in the previous mixture, the percentage of fat being raised and the number of calories per kilogram of body weight somewhat higher. The results of the analysis of this mixture illustrate very well how necessary it is to know the composition of infants' food in percentages of the various food elements and how little physicians really know what changes they make in the composition of a food when they add or leave out a little of this or that ingredient, unless they figure it out in percentages. I am sure that the physician who made these changes in this food would be amazed if he was shown what he really did by them.

What would be expected to result from these changes happened. The baby did not want all her food. The constipation remained the same, she gained but little, the eruption on her face increased, and also appeared on the buttocks and neck.

After a week, milk of magnesia was used in the mixture in place of the lime-water. The physician probably ordered this change because of the constipation. Two ounces of milk of magnesia is a very large dose, however, for a baby of thirteen weeks. One or two teaspoonfuls a day is ordinarily enough to give several movements. It should be given all at one time in one feeding, moreover, instead of being mixed with the food for

the whole day, so that the amount can be varied as necessary. It may be that the physician had been reading some of the advertisements of the manufacturers of milk of magnesia or had just been visited by one of their agents, and, believing what he had read or been told, gave it to "prevent the formation of dense curds in the stomach, render the milk more digestible, and overcome any tendency to constipation, biliousness, or milk dyspepsia." If so, he rather overdid it, as the manufacturers claim that "a teaspoonful of milk of magnesia will produce as soft and flaky a curd in milk as 6 ounces of lime-water." The magnesia caused, of course, looseness of the bowels, and the mother went back to the lime-water. The constipation returned and has persisted. The stools are, according to the mother, hard, light yellow, and smooth. The eruption has continued to be about the same. The baby has gained a little in weight, now weighing, at fifteen weeks, 10 pounds.

Our study of her feeding history justifies, therefore, our original supposition that she had been getting too high a percentage of fat in her food. It also shows that, theoretically at least, she has been given as one of the results of this high percentage of fat a food somewhat high in its caloric value. It does not prove, however, that the high percentage of fat in the food is the cause of her symptoms. As these symptoms are those characteristic of an excess of fat, and no other cause for them has been found, it seems reasonable to believe, however, that they are due to the high percentage of fat. Whether they are or not can be easily determined by cutting down the percentage of fat in her food and giving her a more reasonable mixture.

When babies have a disturbance of the digestion or of nutrition from an excess of fat in the food, it is always advisable to cut the percentage of fat way down at once, rather than to cut it down a little at a time until the tolerance is found. Time is always saved by cutting the fat out entirely, or almost entirely, and then finding the limit of tolerance on the way up instead of on the way down. This baby has not vomited and has had no marked disturbances of nutrition on 5.32 per cent. of fat. It is probable, therefore, that it will not be necessary to cut out the

fat in her food entirely. She can, I think, take 2 per cent. of fat without difficulty. She has never shown any signs of intolerance for milk-sugar or protein. There seems to be no reason, therefore, why we should not give her higher percentages of milk-sugar and protein—7 per cent. of milk-sugar and 1.50 per cent. of protein seem reasonable. It will be well to keep on with the three-hour intervals. She ought to sleep through from 10 to 6, but as she is accustomed to being fed twice in the night, we will at first cut out but one night feeding, giving her seven feedings in the twenty-four hours. If we cut out two feedings it would be necessary to increase the amount of food at each feeding so much that she probably could not take it. Four ounces at a feeding would seem to be about the right amount. Seven feedings of 4 ounces will give her 28 ounces in the twenty-four hours. As she has never vomited or shown any symptoms of difficulty in the digestion of casein, there is no reason for adding an alkali to her food. Her mother can get clean milk and is so situated financially that she can keep it cool. It is not necessary, therefore, to pasteurize or boil the mixture. There being no difficulty in the digestion of casein, there is no reason either for boiling it on this account.

In deciding on the composition and amount of the food, we have thus far entirely disregarded its caloric value. I am quite positive that it is sufficient and not excessive, but we ought to know what it is, so that if she does not gain we shall know at once whether it is because she is being underfed or not. Calculating in the same way as before, we find that 28 ounces of this mixture, which contains 2 per cent. of fat, 7 per cent. of sugar, and 1.50 per cent. of protein, gives about 450 calories, or approximately 100 calories per kilogram of weight. This is somewhat lower than the average caloric needs for her age, and as she is underweight, she probably needs more than the average. If she does not begin to gain very soon, therefore, we must give her more nourishment in some way. This may be done by increasing the amount of the food, by increasing the percentage of protein or by adding starch to the food. Probably it will soon be possible to also increase the percentage of fat.

We must now tell the mother how to prepare the baby's food, so that it will contain the percentages of the different food elements upon which we have decided. I will not take up the different steps of the calculation in detail because you are all familiar with them, but will figure it quickly for you. Using gravity, or 16 per cent. cream, $\frac{2}{16}$ of 28 ounces, or $3\frac{1}{2}$ ounces, will give us the 2 per cent. of fat required. The $3\frac{1}{2}$ ounces of cream in the 28-ounce mixture will give us $\frac{3\frac{1}{2}}{28}$ of 3.20 per cent. of protein, or 0.40 per cent. As we want 1.50 per cent. of protein, and have thus far only 0.40 per cent., we must get the other 1.10 per cent. from the skimmed milk, which we calculate to be fat free, although we know that it really is not. $\frac{1.10}{3.20}$ of 28 ounces, or $9\frac{1}{2}$ ounces, of skimmed milk will give us the rest of the protein needed. The gravity cream and the skimmed milk both contain 4.50 per cent. of milk-sugar. The 13 ounces of gravity cream and skimmed milk in the 28-ounce mixture, therefore, add $\frac{13}{28}$ of 4.50 per cent., or practically 2.25 per cent. of milk-sugar to it. We want 7 per cent., or 4.75 per cent. more, however, and must add it in the form of dry milk-sugar. 4.75 per cent. of 28 ounces is $1\frac{1}{3}$ ounces. One rounded tablespoonful of sugar is equal to $\frac{1}{2}$ ounce and 1 level tablespoonful to approximately $\frac{1}{3}$ ounce. We will, therefore, add 2 rounded tablespoonfuls and 1 level tablespoonful of milk-sugar to the mixture. 15 ounces of water will be needed to make up the 28 ounces desired. I will now fill out and give her our stock slip, telling her how to prepare and give the food.

Preparation of Modified Milk.—Pour off or, better, dip off with a cream dipper all the cream which is visible on a quart bottle of milk. This is "gravity cream." If there is not enough cream on 1 quart of milk, take off the cream from another quart of milk and mix the cream from the 2 quarts together.

The milk which is left after the cream has been removed is "skimmed milk." Mix as follows:

Gravity cream..... 3½ ounces.
Skimmed milk..... 9½ "
Lime-water..... "
Boiled water ..... 15 "
Barley-water..... "
Milk-sugar..... 2 rounded tablespoonfuls and 1 level tablespoonful.
Heat to.....and keep covered for.....minutes.
Keep on ice.
Give baby 4 ounces at a feeding.
Feed at 6, 9, 12, 3, 6, 9 or 10, and once in the night.

It will probably not be necessary to give the baby anything for the constipation after a few days. In the meantime it will be wise to put a teaspoonful of milk of magnesia, more or less, as the case may be, in the 6 P. M. bottle. Later we will give the baby 1 or 2 tablespoonfuls of orange-juice daily.

The eczema, which is slight, will probably quickly disappear when the food is changed. In the meantime the baby must be kept from scratching, and no water should be applied to the affected areas. They can be kept clean with sweet oil, mineral oil, or lanolin. It will also be wise to keep them lightly covered with some bland ointment like Lassar's paste without the salicylic acid, zinc oxid ointment, or cold cream.

CLINIC OF DR. LEWIS WEBB HILL

CHILDREN'S HOSPITAL

1. CONGENITAL ATELECTASIS. 2. BRONCHIAL TETANY

CASE I

THE first case[1] I have to show you presents a condition interesting in itself, and also of especial importance and interest as regards differential diagnosis.

This baby was born last night at 7.30 P. M. after a very quick labor before the obstetrician in charge got there. The labor was an unusually easy one and lasted about three hours. No anesthetic was used. The cord was around the baby's neck when born, but apparently did no harm, as respiration was immediately established. He cried once or twice soon after birth, but has cried very little since then. He passed meconium during birth. His weight is 7 pounds, 8 ounces. He was slightly cyanosed at birth, and at 8 P. M., one-half hour after birth, the cyanosis was a little more marked. At 1 o'clock this morning the cyanosis became extreme, so that artificial respiration with a pulmotor was used and oxygen was given. This morning he looked a good deal better than he did during the night, although he was still somewhat cyanotic. The nurse says that the cyanosis becomes less when he cries, instead of more, as might be expected.

Physical examination shows a well-developed, normal looking baby, save for a moderate degree of generalized cyanosis. As you can see, his respiration seems perfectly normal in character and rate. His heart is quite normal; no thrill or murmur. The sounds are of good quality. There is no dulness demonstrable

[1] This case was seen with Dr. George May, of Newton, at the Newton Hospital, whom I wish to thank for the loan of the *x*-ray plates.

over the thymic region, nor can the thymus be felt at the suprasternal notch. The cyanosis is not increased, nor is the respiration made difficult by retracting the head. To percussion the right axilla is very slightly less resonant than the left, but there can be detected no change in the respiration on either side, which is quite normal in character. The abdomen, throat, and extremities are negative. The fontanel is not bulging and the reflexes are normal. The baby cries with a strong, lusty cry, as you can notice, and I think you will agree with me that the cyanosis is not changed one way or the other by crying. The rectal temperature is 99.4° F.

Discussion.—We are confronted with a cyanotic newborn baby, twelve hours old. The cyanotic babies represent a rather important group in considering diseases of the newborn, and the differential diagnosis may in some cases be rather difficult. Let us see what are the main causes of cyanosis in the first few days of life:

1. Ordinary asphyxia.
2. Congenital heart disease.
3. Enlarged thymus.
4. Cerebral hemorrhage.
5. Sepsis.
6. Pulmonary atelectasis.

In this case the *ordinary type of asphyxia* from intra-uterine causes can be ruled out, as there was no difficulty in resuscitating the baby after delivery, and he breathed well. Furthermore, the condition of cyanosis has lasted too long to be classed under the heading of cyanosis from the ordinary type of asphyxia.

Congenital heart disease is one of the most common causes of cyanosis in such a baby as this. Although in this case there are no murmurs, it is quite possible to have a congenital heart lesion severe enough to produce cyanosis, and perhaps kill the child, without being able to detect anything abnormal about the physical examination of the heart. Particularly if the lesion is an open foramen ovale there may be cyanosis without a murmur. In pulmonary stenosis, defect of the interventricular septum, or patent ductus arteriosus there is practically always a murmur,

although if the patent ductus is very large and much dilated the resistance to the flow of blood may be so small that no murmur is produced.

In this case it is not possible, therefore, to rule our definitely a congenital heart lesion, although the lack of abnormal physical signs on examination of the heart makes it most unlikely.

In my experience *enlargement of the thymus gland* is not a frequent cause of cyanosis in newborn babies. If in this case the trouble were due to enlarged thymus, it is probable that the baby would have more difficulty in breathing, from pressure, and as you can see, he seems to have little actual difficulty, although his respirations are rather feeble. Percussion of the thymus is very unreliable, and I do not believe that it is possible to detect any except very extensive enlargement of the thymus by this means. With enlarged thymus, retraction of the neck often caused increased cyanosis and difficulty in breathing, owing to increased pressure upon the trachea. It would seem that thymic enlargement is not a probable cause of the difficulty in this case.

Cerebral hemorrhage is one of the commonest causes of cyanosis in newborn babies. It is, of course, most likely to occur during a long hard labor, but may occur in any sort of labor. There are practically always pressure symptoms of some sort, such as exaggerated knee-jerks, spasticity of the extremities, twitching, bulging fontanel, etc., associated with the cyanosis and feeble respiration, and as none of these signs are present in this baby, cerebral hemorrhage need not be seriously considered.

Any newborn baby who is *septic* may be cyanotic from general toxemia. There is no focus of sepsis to be found in this baby; furthermore, he is too young to have developed any septic process. The normal temperature does not help one way or another, as the temperature mechanism in newborn babies is so unstable that it is not uncommon to have a normal temperature with severe sepsis.

Congenital pulmonary atelectasis is a fairly frequent cause of cyanosis in the newborn. *In utero*, inasmuch as the fetus does not use its lungs, they are in an unexpanded condition. With the first few cries after birth the lungs normally become expanded.

Certain feeble babies, especially prematures, may not cry with sufficient vigor to expand the entire lung and there may remain large or small areas of unexpanded lung, with collapsed alveoli (atelectasis). These areas of atelectasis are most likely to be found near the bases behind, but may occur in any portion of the lungs. The physical signs are variable; if the area of atelectasis is large, there may be dulness, with a few fine râles and bronchial or bronchovesicualr respiration. In many cases there may be no physical signs at all, however, and the diagnosis has to be made by the symptoms. The condition may last for weeks, and the chief manifestations of it are asthenia, feeble cry, poor nursing, usually but not always cyanosis, and sometimes subnormal temperature. Convulsions are not at all infrequent, and feeble babies often die suddenly without apparent cause, the condition of atelectasis being discovered only at autopsy.

As regards this particular baby, the most probable diagnosis is atelectasis, partly on account of the symptoms, partly on account of the slight dulness in the axilla, and especially on account of being able to rule out the other causes of cyanosis in the newborn. Congenital heart disease cannot be excluded absolutely, but is unlikely on account of the lack of a murmur.

Prognosis.—In this particular baby the prognosis ought to be good, as he is a robust baby of good weight. The lack of subnormal temperature and the comparative ease with which he breathes are also favorable signs, although he had considerable difficulty last night.

Treatment.—The treatment of an atelectatic baby is to make him cry and to keep him warm. So let us direct the nurse to make him cry by pinching or otherwise irritating him every hour. If this does not suffice we will have him dipped alternately into cool and warm water, although she probably can get him to cry without this. Inasmuch as he is in a hospital we can get a roentgenogram of his chest taken today which should confirm our diagnosis; then we can take another one tomorrow or next day to see what progress the lung is making toward expansion.

Two Days Later.—The nurse reports that she had no difficulty in making him cry and was able to do so every hour.

There is at present, as you can see, only a very slight cyanosis, he is breathing much better, and took the breast this morning without any difficulty. The *x*-ray plates are most interesting

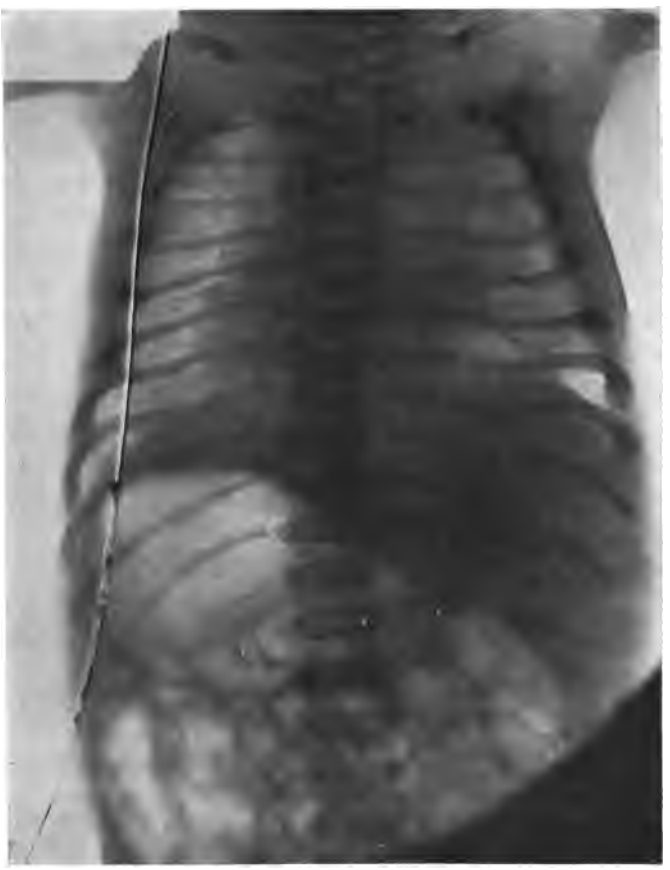

Fig. 58.—Showing unexpanded lung.

and show very satisfactorily the difference before and after expansion of the lung.

Figure 58 was taken shortly after we saw the baby the day before yesterday, and, as you can see, on one side the lung is almost entirely unexpanded, as shown by very definite shadows.

Figure 59, which was taken this morning, shows the lungs almost completely expanded. The *x*-ray is, of course, a most

Fig. 59.—Showing lungs almost completely expanded.

valuable aid in the recognition of congenital atelectasis, but, unfortunately, is not practical to employ except in a hospital.

CASE II

This girl, Mary J., seven years old, entered the hospital some weeks ago with the following history.

Family and Past Histories.—Not important.

Present Illness.—A little over a year ago she had an unusually severe attack of measles, which lasted several weeks. About a month following the measles she began to have attacks of coughing, during which she became blue in the face and extremely dyspneic. The attacks varied a good deal in frequency; some days she would have eight or ten a day, and then would be entirely free from them for a few days. After a month the attacks became more severe and had associated with them stiffness of the body, staring of the eyes, and tonic spasm of the hands and feet. She has had these attacks of spasm of the hands and feet and stiffness of the body without the cough and dyspnea, but the two usually go together. During an attack she has great difficulty in breathing, and her mother says she has been afraid several times that she would choke to death. Each attack usually lasts about ten minutes. Between attacks she breathes without difficulty, has no cough, and apparently feels perfectly well. The condition started in the fall, grew progressively worse during the winter, but in the summer she was much better, although not entirely free from attacks. This winter she has been worse than ever, and some days has had as many as thirty spasms in the twenty-four hours.

Physical Examination.—The essential parts of the physical examination and the laboratory findings are as follows:

A fairly healthy looking, well-developed girl.

Lungs.—Many wheezy râles scattered throughout the chest. No dulness or emphysema. No bronchial breathing. D'Espine's sign absent.

Thymus.—No enlargement made out.

Heart.—Not remarkable.

Chvostek's Sign.—Strongly positive in the first degree, that is, the whole side of the face twitches when the skin is tapped over facial nerve.

Trousseau's Sign.—Positive.

Peroneal Sign.—Positive.

Temperature 99.5° F. Respirations 25.

Von Pirquet slightly positive. White blood-count 28,400.

The night after admission she had eighteen spasms, lasting usually about five minutes each.

The spasms somewhat resembled pertussis except that there was not the crescendo character usually seen in pertussis. The dyspnea started as soon as she began to cough. During each attack she grew very cyanotic, and, on the whole, they were very alarming attacks to watch. When the spasm was finished there was sometimes, but not always, a "whoop," which much resembled that of pertussis or of laryngismus stridulus. The hands and feet were held during most of the attacks in the characteristic position of tetany.

For the first few days she had between thirty and forty spasms in the twenty-four hours. Once or twice she vomited after a spasm; once had a nosebleed, and twice incontinence of feces. It was surprising to see how little the spasms seemed to bother her; between spasms she did not appear at all exhausted, looked well and apparently felt well, save for a moderate amount of wheeziness. During an attack she did not lose consciousness nor did she have at any time what could be called a general convulsion.

The diagnosis of "bronchial tetany" seems justified, as she has the Chvostek sign, carpopedal spasm, etc., and the electric reactions, which have been done, show the characteristic lowering of the threshold of nerve excitation.

She has been here for some time, as you can see by her chart, and is gradually getting much better, having now only three or four mild spasms in the twenty-four hours. Whether her improvement is due to the special treatment she has received, or whether it is simply because she has been kept in a quiet place in bed it is hard to say.

She has been treated as follows:

1. Calcium lactate, gr. 10 every three hours.

This had apparently no effect on the number or severity of the spasms, so was discontinued after about ten days' trial.

2. Parathyroid extract, gr. 1 three times a day, was also tried for about ten days, without any apparent benefit.

3. It was noticed that she excreted very little urine, hardly ever more than a third of the fluid intake. It was thought that she might be retaining sodium chlorid, and that variations in fluid intake might make some difference in the amount of sodium chlorid excreted, and in the number of spasms, so her fluid intake was kept at various levels for periods of several days each—16, 45, and 70 ounces. Changing the fluid intake seemed to have very little effect upon either the number of the spasms or upon the output of sodium chlorid. She had no more spasms when drinking only 12 ounces of water daily than when she took 70 ounces. On two successive days she was given 5 grams of sodium chlorid to see whether the number of spasms would be increased. No increase was noted.

We had about made up our minds to try subcutaneous injections of magnesium sulphate, as advocated by Berend, when she began to improve, and at the present time is having only three or four mild spasms in the twenty-four hours.

Discussion.—This condition was first described by Lederer in 1913, who gave it the name of "bronchial tetany." Curschmann soon after described certain other cases of a somewhat different type from those of Lederer. Short papers by Rietschel and by Wieland appeared in 1913 and 1914, but aside from this very little has been written regarding it. Lederer saw 58 cases of spasmophilia among 5903 ambulatory and 767 ward patients; 6 of these showed "bronchial tetany." His patients were all under six months old and all of them died. In most of his cases there was continued spasm of the bronchioles probably lasting until death, manifested especially by areas of pulmonary atelectasis, cough, rapid respiration, and cyanosis. Two of his cases lived for between two and three months after the onset of symptoms, and more closely resembled the case that we have been discussing. According to Lederer, the condition most likely to be confused with bronchial tetany in little babies is pneumonia. *x*-Ray of the chest is of the greatest value, and a number of Lederer's cases were first thought to be pneumonia until *x*-ray examination showed that the lung condition was one of atelectasis. The atelectasis is caused by tonic spasm of the

bronchial smooth muscle, which shuts off the air supply to the alveoli supplied by that particular bronchial tube.

Since the condition of spasm may last for days, weeks, or even months, the air in the shut off alveoli is absorbed, the walls collapse, and that part of the lung becomes atelectatic. In Lederer's cases there was dulness over certain portions of the lung, which seemed sharply demarcated, while over the rest of the chest there was likely to be emphysema.

In 4 of his cases (the most severe ones) the bronchial spasm was continuous, in the other 2, which lived longer, it was apparently intermittent. Lederer says, "There will probably be reported, as people begin to recognize this disease, milder cases in which the bronchial spasm may last only a short time."

Curschmann noticed that in certain adult cases he had been calling bronchial asthma there was a considerable irritability of the facial nerve, with a positive Chvostek's sign. This led him to suspect that these might be, in reality, cases of tetany. His cases resembled closely the one we have been discussing, with the exception that the attacks were not so frequent. He reports very good results after treatment with calcium lactate. According to Curschmann, atelectasis need occur only in the most severe fatal cases, and there may be all gradations between the severest bronchial spasm and less severe and more temporary spasm, with consequent variation in the clinical picture.

In this girl the bronchial spasm is certainly not continuous, and the larynx is probably involved as well, as shown by the "whoop" which sometimes occurs. It is, of course, quite impossible to tell how much of the difficulty with respiration is due to bronchial and how much to laryngeal spasm.

Inasmuch as the spasmophilic attacks started soon after she had measles, it is not unreasonable to suppose that the measles may have had something to do with it, in just what way, it is not certain. It is the same condition as spasmophilia in little babies, and probably has something to do with an insufficient calcium retention, the same sort of process that occurs in babies with spasmophilia. Coincident with this there is probably an overstoring of sodium in the body, which increases nerve ex-

citability. Disease of the parathyroid glands has also been found in a few cases of spasmophilia, but is by no means constant. It is not difficult to understand how babies who are having trouble with their feeding should have a disturbance of the salt metabolism, but it is hard to see why a well-grown, healthy girl such as this should develop the condition. It is interesting to note that feeding her sodium chlorid by mouth did not seem to increase the number or severity of the spasms. Large doses of calcium chlorid or of calcium lactate sometimes work very well in spasmophilia. As near as we could tell with this girl they seemed to do her little good.

The condition of bronchial tetany is a most unusual one, and for this reason I report it, as there is practically nothing concerning it in the American literature.

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CLINIC OF DR. EDWIN T. WYMAN

CHILDREN'S HOSPITAL

TWO CASES OF ACQUIRED HEART DISEASE IN CHILDHOOD

I AM going to show 2 cases of acquired heart disease, discuss their diagnosis and treatment, and briefly take up other types of the disease.

Case I.—M. G. is an only child, eight years of age, whose parents are still living and well. He was born at full term after a normal labor and was breast fed for ten months. He had bronchopneumonia when two years old and at three years of age had measles.

This winter he has been attending a military school. Three weeks ago he was admitted to the school hospital, with what was thought to be a mild attack of influenza; as he was not very sick, at the end of a week he was discharged from the hospital and came home on a vacation. On leaving the hospital he did not seem sick and was not feverish, though for the past week he has been complaining of a slight pain and stiffness in his ankles. After he got home the pain and stiffness in his ankles was more severe and he began to complain of pains in his wrists and elbows. He was allowed to be out of doors as usual until two days ago, when he seemed to have considerable fever and his left knee and ankle became slightly swollen so that he walked with difficulty. On admission to the hospital his mouth temperature was found to be 102° F.

Physical Examination.—He was well developed, well nourished, of good color, and did not seem very sick, although he complained of a great deal of pain in his left ankle and his left knee and slight pain in his right ankle and both elbows when

they were moved. The left ankle and knee were slightly swollen and were tender on pressure, and there was a slight redness and heat of the left ankle. The other joints were not tender. The tongue was clean. There were two carious molar teeth. The tonsils were moderately enlarged, showing some evidences of previous infection. There was no enlargement of the peripheral lymph-nodes. The cardiac impulse was visible and palpable in the fifth space 8½ cm. to the left of the median line. The upper border of the relative cardiac dulness was at the lower border of the second rib. The left border was 8½ cm. to the left of the median line, the right border 2½ cm. to the right of the median line. The cardiac action was somewhat irregular and the pulse-rate was 120. The first sound at the apex was good, but continued into a short, blowing murmur, transmitted into the axilla. The second sound at the pulmonic area was accentuated and there was no venous hum in the neck. The murmur did not disappear on changing the position of the patient from lying to sitting. The lungs were normal to auscultation and percussion, the abdomen was level, there were no masses or tenderness, and no spasm. The spleen and liver were not palpable. The extremities were normal. There was no spasm or paralysis. The knee-jerks were equal and normal. The hemoglobin was 90 per cent. (Sahli). The leukocyte count was 12,000, and 85 per cent. were polynuclears. Examination of the blood-smear showed nothing abnormal. The examination of the urine was normal.

Discussion.—The history and condition found in the joints are typical of rheumatism in childhood. There is undoubtedly something abnormal about the heart; with the rheumatic infection, one would be on the lookout for cardiac involvement, as unfortunately the heart is frequently involved with this type of rheumatism. In this case a functional murmur can at once be excluded on the good color and the absence of a venous hum in the neck. The enlargement of the heart and the absence of a murmur at the pulmonic area are also against it. There is probably some involvement of the myocardium, as there nearly always is with rheumatic infection of the heart, but any marked

degree of myocarditis can be ruled out by the character of the impulse, the strength of the first sound, the accentuation of the second pulmonic sound, and the absence of enlargement to the right. The increased pulse-rate would indicate slight myocardial involvement. The murmur is characteristic of an early endocarditis of the mitral valve. The diagnosis is, therefore, acute endocarditis with some involvement of the myocardium.

Prognosis.—There is no immediate danger of life from the endocarditis. If proper treatment is carried out, and a recurrence of the disease with further damage to the heart prevented, the heart will not be seriously affected. There is very little chance of complete recovery, as there is almost certain to be a permanent deformity of the mitral valve. The exact extent of the myocardial infection and the later cicatricial change in the mitral valve can only be told by later observation.

The prognosis may be said to depend chiefly on the possibility of preventing a fresh infection. The power of recovery in youthful hearts is much greater than in adult life, but the prospects of a reinfection with further damage to the endocardium and myocardium are much greater in youth than in adult life.

Treatment.—The most important thing in the treatment is rest, everything else is subordinate. He should be kept in bed for four or six months, or longer if necessary, in the hope of rendering the heart functionally sound. The persistence of a mitral murmur or slight dilatation does not preclude the possibility of securing this result. A week in bed at this time may mean a year of life later on; he must be kept in bed not only during the acute stage, but for months longer. If the temperature ceases to be elevated we may conclude that the active general infection is gone. It is desirable to maintain the absolute rest in bed as long as any signs of active endocarditis persist; of these the most useful is an increased heart rate and dilatation of the left ventricle. The most satisfactory sign of recovery is the slowing of the pulse-rate. If the rate continues above 100 and no other cause can be found, we may assume that the infection is still present and further rest is required. When

the pulse-rate is reduced to 90 the patient may be allowed to sit in a reclining chair; previous to this, while the patient is still in bed, he may be gradually accustomed to elevation of the head by the addition of an extra pillow for an hour or more daily. Greater freedom may be permitted when it is found that with the increased exercise the heart-rate is kept below 100. When he is allowed out of bed the change should be only from bed to sofa or easy chair by degrees. Later a little walking about the room may be permitted, careful observations being made as to the effect of exercise on the heart, and more especially to the marked increase of the pulse-rate, and of breathlessness or fatigue on walking. His life must be most carefully regulated for a year or two after he gets up, and the amount of exercise limited. He will probably feel well and wish to do what other children do, but he must, however, be restrained, and his whole life planned to save his heart and to prevent reinfection.

It is often said that salicylates are useless in the treatment of endocarditis, but this is going too far. As regards the direct effect of salicylates on myocardial or valvular inflammation, the result may not be appreciable or manifest, but we have to consider the general rheumatic infection which had started the endocarditis and which tends to keep it up, and if we keep this infection under or remove it from the system by means of salicylates, we are undoubtedly improving the prospects of the patient as regards his heart. It seems reasonable to me that if the salicylates help the rheumatism they will have a very favorable influence on the endocarditis, which is a manifestation of rheumatism. It is hard to understand at any rate how they can do harm in rheumatism as some writers think they do. The most satisfactory preparation of salicylic acid is aspirin. This boy during the acute stage and until his joint system and fever are relieved should have 5 grains every four hours unless he gets toxic symptoms. If he does, the dose should be reduced and he should be given what he can stand. His compensation is perfectly good. There is, therefore, no call for either cardiac stimulants or tonics. It is often said that if the pulse is weak

we must stimulate the heart; but an inflamed heart is more in need of rest than stimulation. The less stimulation of the heart that is attempted during the stage of acute endocarditis the better it is for the patient. If he is restless or uncomfortable he may be given bromid of sodium or potassium in 5- to 10-grain doses, or morphin in doses of from $\frac{1}{16}$ to $\frac{1}{8}$ grain. By securing physical rest and sleep, and by removing all nervous disturbances, and the reflex cause of disturbance we can do a great deal to secure the maximum of rest to the heart.

There are no special indications as to his diet. At first he must be given milk and a starchy diet. If he does not take his food well, milk-sugar can be added to his milk up to 7 per cent. to increase the caloric value. Later there is no objection to meat, eggs, and vegetables. Special attention must be paid to his nutrition, and it must be seen that he gets a sufficient number of calories, about 1325, as the condition of his heart muscles depends to a certain extent on his general nutrition. After he has been up and about, all precaution should be taken against a recurrence.

From a preventive point of view I think it is advisable to give him a course of salicylates, especially during the winter months. One week every month he should take 5 grains of aspirin three times a day. If it is possible he should spend his winters for a few years in a mild climate, and if this is not possible he should be protected by warm woolen underclothes and proper footwear from the cold damp weather during the winter months to guard against the risk of fresh rheumatic infection.

As soon as he is sufficiently recovered, his carious teeth should be filled or they should be extracted if there is any evidence of abscess formation. His tonsils should be enucleated, as they now show evidence of disease. The tonsils or teeth may be a source of the rheumatic infection, and these sources of infection should be eliminated as soon as possible. This should not be done until the acute symptoms have subsided, not for ten or twelve weeks at any rate, as there is always the danger of starting up a fresh infection.

If, however, the acute symptoms do not quiet down after a

month or six weeks, and the heart continues to get progressively worse, and if the tonsils are diseased or show evidence of subacute infection, I believe a tonsillectomy should be done even during the acute endocarditis. In these cases the tonsillectomy, removing the source of infection which is keeping up an active endocarditis, will often be followed by marked improvement.

It is frequently very difficult to tell on inspection whether or not the tonsils are diseased.

The small, submerged, apparently healthy looking tonsil on pathologic examination may prove markedly diseased. Considering the fact that the rheumatism and endocarditis are in a great measure results of diseased or infected tonsils, I firmly believe that, as a routine prophylactic measure, every child who has had an acute endocarditis should have the tonsils enucleated if there is any question at all of their being the source of the infection.

It is of the greatest importance when the tonsils are removed that they be completely enucleated, for if small tabs of tonsillar tissue are left, the focus of infection will probably still be there and the benefits derived from the operation will be nil.

Case II.—G. R. is an only living child, eleven and a half years of age, whose parents are living and well. One child died soon after birth. There was nothing in the family history to suggest syphilis. There was no tuberculosis in the family and he had had no known exposure to it. He was born at full term after a normal delivery; his birth weight was 7½ pounds. He was breast fed for three months, then put on a whole milk modification, and did very well as a baby. He was very well until four and a half years old, when he had an illness associated with pains in his extremities, which was treated for "pin-worm fever." He was in bed only for about two weeks, though the pains and swelling in his joints persisted for about six weeks. The following summer he played about with other boys, but it was noticed that he got short of breath easily, and oftentimes at night his feet and ankles would be swollen. It was also noticed that his abdomen was growing larger. When

he was seven and a half years old he was admitted to the hospital. The examination of the heart at that time showed the upper border of cardiac dulness at the upper border of the third rib. The right border was 4 cm. to the right of the median line, the left border was 12 cm. to the left of the median line. The action was regular, the pulse-rate 120. The cardiac impulse was heaving; it was seen and felt in the fifth and sixth spaces 11 cm. to the left of the median line. The first sound at the apex was somewhat feeble and was followed by a loud blowing murmur which was transmitted to the axilla and back. The second sound in the pulmonic area was louder than the second sound in the aortic area and was accentuated. The lower border of his liver was palpable 9 cm. below the costal border in the nipple line. There was flatness in the flanks and the area of the flatness changed with change of position and there was a definite fluid wave. There was some edema of the legs and he was somewhat cyanotic and had a good deal of dyspnea. The abdomen was tapped and 4½ quarts of serous fluid removed. The urine was normal. He was given tincture of digitalis, 10 minims every eight hours, and theocin, 5 grains three times a day. The edema disappeared and he was discharged from the hospital in about six weeks.

The next two years of his life were carefully regulated, and his heart was stimulated from time to time with digitalis. He had some edema of the extremities, and always had signs of fluid in the abdomen, though not enough to necessitate tapping. Since that time, about three years ago, up to the present time he has had very little medical care, and now is able to walk about, although upon any exertion he has a good deal of shortness of breath, and after he remains on his feet any length of time his ankles will become a little swollen.

Physical Examination.—He was well developed, well nourished and of good color, unable to lie down without much discomfort, conscious, mentally clear, no edema of the face or chest, and no enlargement of the superficial veins of the chest. There was a marked pulsation of the veins of the neck. The tongue was clean, the teeth were well cared for and in healthy

condition, the tonsils moderate in size, with no inflammation. The cardiac impulse was visible and palpable in the midaxilla. Systolic retraction could be seen in the fourth and fifth interspaces to the left of the sternum. The upper border of relative cardiac dulness was at the second interspace, the right border 5 cm. to the right of the median line, the left border 16 cm. to the left of the median line. There was a loud systolic murmur over the entire precordia, heard best at the apex, and transmitted over the entire chest. There was a presystolic murmur and presystolic thrill at the apex. The second pulmonic sound was louder than the second aortic sound and was loudly accentuated. The cardiohepatic angle was acute. The action of the heart was regular. The pulse-rate was 108. There was no Corrigan pulse and no pistol shot in the groin.

The lungs: There was dulness changing to flatness toward the base on the left side below the spine of the scapula, the respiration and voice sounds in this area being somewhat diminished in intensity and more of a bronchial character. The vocal fremitus over this area was somewhat increased and medium moist râles were heard over this area. The abdomen was distended. There was a marked flatness in the flanks and hypogastrium, the upper border of flatness being concave as he lay on his back, with the area of flatness changing with change of position, and there was a definite fluid wave. No masses were felt. The umbilicus was slightly protruding. The upper border of liver flatness was at the upper border of the fifth rib in the nipple line. The lower border of the liver was palpable 12 cm. below the costal border in the nipple line. There was a slight edema of the lower legs and ankles. There was no enlargement of the peripheral lymph-nodes. The Von Pirquet test was negative. Hemoglobin was 85 per cent. (Sahli). Red corpuscles 5,200,000. Leukocytes 7500. Polynuclears 78 per cent.; mononuclears 22 per cent. The smear showed nothing abnormal. The urine was normal.

Discussion.—The most reasonable explanation of this boy's condition is as follows: The illness, which was called pin-worm fever, was in all probability rheumatism. He developed an en-

docarditis, myocarditis, pericarditis, and mediastinitis which resulted in a badly damaged mitral valve and in the obliteration of the pericardial cavity and the formation of adhesions between the pericardium and the mediastinal tissues.

The heaving impulse, the accentuated second pulmonic sound, and the double murmur at the apex show that there is a lesion at the mitral orifice, both an insufficiency and stenosis. The points in favor of the assumption that he has an adherent pericardium and mediastinitis are the marked enlargement of the heart, the apex lying in the axilla, and the right border extending well to the right of the sternum, more hypertrophy of the heart than would be expected with the mitral lesion. The systolic retraction in the intercostal spaces, the diastolic collapse of the veins of the neck, and the enlarged liver and ascites seem sufficient to justify the diagnosis. The negative tuberculin test shows that this process was not tubercular, as it sometimes is.

The hypertrophy of the heart and pleural adhesion and thickening account for the signs in the back. The pleural adhesions, the pressure of the distended abdomen, and of the enlarged liver interfere with the expansion of the lungs and cause a congestion at the bases, which accounts for the râles.

The chronic adhesive pericarditis has produced a cirrhosis of the liver due only in part to passive congestion. The edema of the legs is probably not due to the passive congestion, but to the pressure of the fluid in the abdomen on the inferior vena cava.

The diagnosis is, therefore, chronic valvular disease of the heart and chronic adhesive pericarditis.

Prognosis.—He may live for months or even years, but eventually the additional burden imposed on his circulatory apparatus as he grows older will lead to a fatal ending. The outlook is hopeless except for prolonging his life.

Treatment.—Tapping the abdomen from time to time will make him much more comfortable. Other treatment must be mainly rest and digitalis. He should have an easily digested diet, as his digestive system is apt to be disturbed by the results of venous stasis. His bowels should be kept open with saline cathartics.

In other mild cases of rheumatic infection of the heart the endocarditis or myocarditis or even a pericarditis may subside without leaving any permanent damage. This is the result always aimed at in our treatment, and most frequently secured after the first attack. With each successive reinfection the chances of a complete cure become less and less.

The signs of a slight lesion are unfortuntely not always clear, but certainly in early life one can often observe the gradual disappearance of a systolic murmur thought to be due to a lesion of the mitral valve or to cardiac dilatation which appeared to be due to myocarditis after a definite infection. A systolic murmur heard in the precordial region is not evidence of heart disease and taken by itself is never an indication for treatment. It is not going too far to say that by many the diagnosis of heart disease has often been made simply on the discovery of a cardiac murmur. It may have been functional, organic, congenital, or extracardiac, but simply because a cardiac murmur had been discovered, the child was pronounced to have "heart disease." The discovery of the cardiac murmur was followed by extreme precaution lest something further should develop, and these children have been unnecessarily treated as invalids.

It is often difficult to differentiate between organic and functional murmurs. There are, however, some characteristics of organic murmurs, particularly mitral insufficiency, which help in their differentiation. In organic disease there is some degree of cardiac enlargement and accentuation of the pulmonic second sound. Organic murmurs are usually persistent under varying positions of the patient, lying and sitting, during rest and after exercise, and from day to day. They usually persist during respiration and when the breath is held. They often have a musical element or other characteristic tone which is not heard in the case of functional murmurs. Organic murmurs have a wider range of conduction toward the axilla and back, and one or the other sound of the heart may be faint. Practice alone will enable one to say in difficult cases whether a murmur is functional or organic. It is always advisable to consider carefully the other symptoms of the disease which may be present,

and in no case is it justifiable to diagnose endocarditis on the evidence of a doubtful mitral systolic murmur. A presystolic or diastolic murmur can be safely considered as being of organic origin.

The valvular murmur of organic disease is important chiefly from the diagnostic point of view. It is an indication of a valvular lesion that an endocarditis is, or has been, present. It must necessarily be considered along with other signs of rheumatism and in itself is not usually of any prognostic value. So that, in seeking to determine the significance of a valvular lesion as evidenced by a murmur, we have to consider what effects, if any, the lesion produced on the heart and circulation. These will be found in some cases in the form of dilatation, hypertrophy, cyanosis, and dyspnea. Even in the presence of such symptoms care must be taken lest in ascribing the symptoms to a valvular lesion one has overlooked associated changes in the myocardium.

Valvular organic murmurs in childhood are associated chiefly with the mitral and aortic orifice, lesions of the pulmonary and tricuspid valves being extremely rare in connection with acquired disease. It is of the greatest importance to make an early diagnosis if good results are to be obtained, for in many cases the patient will have no symptom referable to his heart until the symptoms of heart failure appear, and then the damage to the heart is beyond repair.

While streptococcus, staphylococcus, pneumococcus, and gonococcus may produce a heart infection, and less frequently, the tubercle bacillus, the typhoid bacillus, the anthrax bacillus, the colon bacillus, the diphtheria bacillus, the Bacillus pyocyaneus, and bacillus of influenza, the vast majority of cases of organic heart disease in childhood are due to rheumatic infection. In childhood and youth we are dealing with the early stage of heart disease which is of an inflammatory nature as contrasted with the heart disease of later life which is degenerative and fibrotic in character. The cardiac irregularities do not bear the same outstanding position in the symptomatology of heart disease that they do in adult life.

Chorea and heart disease are often associated, and experience

has taught us that both have a common origin in rheumatic infection. Yet they may be found separately or in combination in patients who give no history of having suffered from definite rheumatic fever.

The frequency of rheumatic infection after the age of four years throughout the whole of childhood demands careful treatment and frequent examination of the heart, in all cases presenting any of the symptoms of rheumatic infection which may be manifested by a definite attack of rheumatic fever, chorea, tonsillitis, slight pain in the limbs, the so-called "growing pains of childhood," or a slight arthritis. While these rheumatic symptoms may come and go, whether treated or not treated, and leave no trace behind, there is always danger of cardiac involvement; and careful observation and treatment, in cases presenting these symptoms, will probably be effective in saving the heart from infection or at least from permanent damage.

In the class of cases in which recurrent attacks of rheumatism are taking place, after the teeth, tonsils, and alimentary tract have all been examined and put into as healthy a state as possible and the child's life carefully regulated, one can but assume that in these cases there is some focus of infection in the body which cannot be detected by our present means of investigation, which constantly or at intervals pours out a fresh supply of organisms. The essential part of the treatment is to combat the infection by salicylates and change of residence to a warmer and drier climate. In addition, I think, from a preventive point of view, it is advisable to give a course of aspirin to be taken for a week or ten days every month for a year or two.

In another class, where a progressive carditis is present, there is a condition of chronic infection of the myocardium. An endocarditis and pericarditis may also exist. On examination of the heart the chief signs of infection are a persistently rapid action of the heart and a tendency to progressive dilatation. In this condition the salicylates are of little use, as the disease has reached a stage of local rheumatic infection as opposed to the blood infection of rheumatism, and the satisfactory results obtained in the latter cannot be expected. A chronic infection like

this tends to die out if we can maintain the normal resistant power of the body at a high level, and the cardiac condition is most likely to improve under rest and freedom from disturbance. The patient should, therefore, have a diet of nourishing food and should be kept in bed for a prolonged period.

Open-air treatment may prove very beneficial. Our therapeutic measures in this condition are limited, but patience in the treatment will often be rewarded with a satisfactory result.

In cases where there is a malignant endocarditis, although there is usually a pathologic basis in proliferative ulceration of the heart valves, the disease is really a form of general septicemia, and is to be treated as such. The outlook in these cases is hopeless, and one should try to maintain the patient's strength and make him as comfortable as possible.

CLINIC OF DR. KARLTON G. PERCY

CHILDREN'S HOSPITAL

CHRONIC INTESTINAL INDIGESTION FROM STARCH SHOWING INDICAN REACTION. REPORT OF 33 CASES

I DESIRE to speak today of a group of 33 cases of chronic intestinal indigestion due to malassimilation of starch, in children from nineteen months to twelve years of age, which have shown an indican reaction in the urine as a persistent sign on first examination, with or without a sugar reaction to Fehling and Benedict.

Our present knowledge of indican has been directly opposed to its appearance in this type of case (unless we can attribute it to starvation, as has been recorded by Müller[1] among the insane, or to retarded peristalsis of the small intestines).

Indican is found in the urine as the basic indoxyl-sulphuric acid $C_8H_7NSO_4$. This is derived from indol which is oxidized in the body from indoxyl, then conjugated with sulphuric acid,[2] and is eliminated as sodium or potassium, indoxyl-sulphate, or indican. It is supposedly derived from intestinal putrefaction of protein. Indicanuria has been produced by local stasis in the small intestines by Joffe,[3] Ellinger and Pintz,[4] but no increase has been demonstrated through obstruction of the large intestines.

C. E. Simon[5] says an increased elimination of indican is seen in—

1. All diseases associated with increased degree of intestinal putrefaction, due to ileus, in carcinoma of stomach, and chronic gastritis.
2. Impeded peristalsis of small intestines.

3. Albuminous putrefaction may also take place within the body in such cases as empyema, large deep-seated abscesses, etc.

The quantitation of indican is too elaborate to be of value, and there are very few statistics on this subject. Joffe's figures in eight observations in normal adults of 6.6 mgm. from 1000 c.c. urine are the only figures I find at hand.

I employed the commonly used Obermeyer's test,[7] at first clarifying with lead subacetate, and finally without clarification, as I found little if any change in the intensity of color by this method.

Five c.c. of urine are mixed with 5 c.c. of Obermeyer's reagent in a stoppered 20-c.c. test-tube. This mixture is shaken for approximately two minutes. Very slight warming increases the rapidity of this reaction; 2 c.c. of chloroform are then added, the resultant mixture shaken two minutes, and then the chloroform is allowed to percipitate and the resulting blue color judged by its intensity as +, ++, or +++. This indican or indigo-blue chloroform mixture may be judged in a colorimeter against known dilutions of Fehling's solution, which it simulates if the basic lead acetate treatment is first used.

A series of 53 cases of children not apparently suffering from chronic intestinal indigestion with starch were given urinary analysis, with 51 negative results and two reactions of only one plus indican.

This series of mine was begun without any apparent scientific reason save that I had often noted in this type of indigestion a highly colored urine probably due to concentration. This type of case gave such strong indican reactions that I wished to rule out concentration as a factor. In 3 separate cases, 3, 17, and 29, I collected twenty-four-hour amounts, then diluted the urines with tap-water to relatively normal outputs for their approximate ages, and still obtained a strong indican reaction. Therefore it seemed to me that this must be a relatively positive indican excess. Further, as the indican rapidly disappeared from the urines as soon as a proper dietary had been begun, I felt it still further a true incident to this deranged

digestive condition. All the cases presented very much the same sort of history. Most of these children were brought to me, not that anything definite was the matter, but rather because they were not up to par.

1. Anorexia was a prominent symptom in 22, or 66 per cent., of this series. This varied from mild disinclination to eat like other children, to an abhorrence to any food at the table. A more careful history generally elicited the fact that the children would willingly eat sweets, starches, raw apples, condiments, and food directly contraindicated to their digestive ability. Five of this series without loss of appetite were seen for a history of convulsions, and 3 of these were normal or above in weight per age and per height.

2. *Underweight* for their age and height appeared as an important factor, not only from the parents' point of view, but from clinical evidence. In 27 cases noted exactly, this occurred in 23 cases, or 85.9 per cent. This varied from 30 per cent. below par to 1 per cent. And I find that only 4 children under par did not fall 7 per cent. or more below, thereby putting them into the class of malnutrition. I used Dr. Thos. D. Woods' chart published by the Department of the Interior at Washington, D. C. Three of the 4 who did not show malnutrition or underweight were seen for petit mal or convulsions of greater severity. Cases 5 and 8 are interesting.

Case 5, a boy of six, came to me because he was considered dull in school and because he had attacks simulating petit mal at table, around the house, and even in the street, when he would "suddenly stop in whatever he was doing, drop what he held, let his eyes roll up, and be far off for a part of a minute." He had enuresis, was a voracious eater, at meals and between, of a diet none too carefully guarded. His physical examination was absolutely normal, and the urine showed a slight sugar reaction immediately on boiling, and a strong indoxyl. Stool was large, foul, fermented and undigested, with starch and cellulose in excess, and a strong fermentation +++ after boiling with dextrose and incubation, denoting the gas bacillus. This patient was put upon the lowest possible starch and thin

milk diet, high in protein, with an absolute disappearance of all his symptoms within three weeks.

Case 8 was even more interesting and instructive to me. An only child, under the best of conditions, except his life in an adoring grandparents' house, where he was made much of by all the many relatives. Breast fed for four months, colicky, but gaining well until three months, when he had a convulsion and was unconscious six hours. At about eighteen months he had a gastro-intestinal upset, with high temperature and convulsions. Physical examination and lumbar puncture said to have been normal. I saw him at thirty-four months again in consultation after three to four weeks of recurrent intestinal upsets, some with and some without convulsions, with dopiness between. At this time he showed a very decided, though improving right-sided hemiplegia and aphasia. His stool was not remarkable and urine not tested for as noted in this series. I felt it was a case of cerebral edema due to gastro-intestinal toxemia, and gave a good prognosis and a relatively stringent diet. I was called again five months later for another series of convulsions, to find a typical history of starch indigestion and its coincident symptomatology. The baby was about normal weight. His stool showed starch in excess and a positive gas bacillus test, and his urine 1019, with retarded Fehling's and ++ indican. Following this examination I asked that he be put on 1 quart of buttermilk a day plus meat, egg, broth, custard, junket, and zweibach for one month. This diet was gradually increased, and though his starches are still low, he has gone eight months with only one slight petit mal following a day of excitement and possible change in diet. I had no way of following up the urinalysis, though I felt certain it followed the same course as the rest of the series.

Fussiness and change of disposition are very noticeable symptoms in this series, recorded in 24 cases, or 72 per cent. These children are whiney, cantankerous, wilful, often very nervous and high strung.

Restlessness at night is another very frequent factor, if sought. These children are light sleepers, tend to roll and toss,

throw off the covers, cry out in their sleep, grind their teeth, and have nightmares of varying severity. This was noted in all but one case in my histories.

Enuresis appears in only 10 of this series, or 30 per cent., though mentally I would have put it higher. Dr. J. I. Grover's excellent work on this subject shows the relationship between enuresis and improper dietary.

Pin-worms were noted in 3, or 10 per cent., of these children. This is of interest, as the usual symptoms attributed to this infection are well summed up in the symptomatology of this deranged condition of digestion, and I feel sure that if more care were taken before a "slap dash" diagnosis of pin-worms was made, the general well-being of chronic indigestion children would be improved.

Convulsions appeared in the history of 6 cases, or 18 per cent. Some occurred in connection with the onset of acute infectious diseases, others without any apparent concomitant cause.

Case 3, a girl of nearly thirteen, was having eight to twelve attacks of petit and grand mal a day. Her parents refused to allow her to go to the hospital for study and she refused a limited dietary and I lost sight of her. Six months later I found her under the care of an associate in the Children's Hospital, who had her on buttermilk and low starch diet, with a 9-pound gain and almost complete disappearance of symptoms.

From the standpoint of *urinalysis* every case but one (and this a specimen over twenty-four hours old) showed acidity, the specific gravity was high, ranging from 1016 to 1030, with an average between 1018 and 1024. This is probably due to the low fluid intake only, as these children dislike milk and forget to take water. This high gravity disappears as soon as the patient begins to eat and drink more.

Albumin was found in only 1 case, and this unaccounted for except as a probable orthostatic albuminuria.

The presence of *sugar* in small amounts is of great interest, though of little significance to me. Rarely was it sufficient to come down immediately, and it was only through lax methods of leaving my test-tubes uncleaned that I noticed it. It showed

both by Fehling and Benedict, though the former is my routine. I found it in 22 cases, or 66 per cent., on the first visit, and its continuance on subsequent visits was an indication of failure to follow the diet subscribed.

A positive *indican* reaction either +, ++, or +++ was present in my whole series, though I have a few cases of chronic starch indigestion which have not shown it, and often numerous cases of acute fermental indigestion without it. This reaction disappears in ten days to seven weeks, apparently coincident with the stage of the indigestion and the care used in following the dietary.

Starch was evident in every case, as shown by the Lugol stain microscopic slide method. Coincidently it is interesting to note a predominance of iodophylic bacteria. The presence of the much berated gas bacillus was sought for in 21 cases. I used Dr. Kendall's revised method of fermentation tube, and accepted the presence of over 1 c.c. of gas at the end of sixteen hours' incubation as a positive gas bacillus test. This has proved sufficient if one is familiar with the technic, as proved from the Out-Patient Laboratory of the Children's Hospital, by bacteriologic examination at the Harvard Medical School.

I will not go into the ever-open discussion of the pathogenicity of this organism, but suffice it to say that, when found, I believe it best removed from the gastro-intestinal tract in the fastest possible time, and this is best accomplished with the aid of ripened lactic acid milk.

The astonishing thing in the history of these cases is that in most every one we hear that the child's bowels "are all right" or need a very slight laxative. As a matter of fact, they are generally unformed, very foul, and often frothy, with undigested particles.

As for my treatment: I have tried to be absolutely scientific and have treated the conditions according to the signs which are present. This has been the giving of the lowest possible starch diet which seemed indicated in each given case. In the presence of gas bacillus, I added lactic acid milk, or occasionally only the cultures or tablets. Frequently I reduced the fat in-

take, if a secondary fat indigestion was demonstrable in the stools. In general, I began with lactic acid milk or skimmed or thin milk, 1 or 2 quarts a day. Meat broths without remaining vegetable fibers. Rare meats, chop, steak, roast beef, lamb, or chicken. Eggs. Custards. Junket. A moderate amount of twice cooked bread or zweiback, and rice jelly as the only starchy cereal. This was to be continued until the child regained his or her appetite and lost many of the fussy nervous symptoms. Sometimes it took one week, more often two weeks, occasionally three weeks. Queerly enough, I have had my best gains in weight on this low caloric diet, often as high as 1 to 2 pounds a week, and in one instance 4 pounds of true weight recorded in my malnutrition class at the Children's Hospital, and not recorded in this series for lack of fuller data. As soon as the digestion warrants I add to this dietary assimilable starches and more fats, not allowing potatoes, raw apples, and any sweet pastry for a number of weeks or months.

Medicinally, I have used nothing as a routine, and very little in any instance. A few cases have had nux or gentian for psychologic as well as physical reasons. A few have needed laxatives. The one type of laxative that has seemed contraindicated has been the fruit laxatives that act mostly by the fermental activity, such as syrup of figs, frutosan, prunoids, etc. The results have been most satisfactory, the symptoms disappear, and the patient gains weight, color, and poise. In 20 cases where I have definite statistics over five weeks in duration the average gain has been 4.33 ounces a week or about 175 per cent. above normal.

Whatever this phenomena of a positive indican reaction in a majority of chronic intestinal indigestion with malassimilation of starch is due to, the fact remains as a matter of interest. I am led to believe that it is in part due to partial retardation of the peristalsis in the small intestine plus a chemical reaction on ingested protein by the action of latent organism in the intestinal tract.

TABULATION OF 33 CASES OF STARCH INDIGESTION

Number and Initials.	Sex.	Age.	Per cent. underweight.	Anorexia.	Fussy.	Restless at night.	Enuresis.	Pin-worms.	Stomachache.	Convulsions.	Vomiting or acidosis.	Reaction.	Specific gravity.	Sugar.	Albumin.	Indican.	Starch.	Gas.	Movements a day.	Weekly gain in weight. Weeks.	Weekly gain in weight. Total gain.	Duration of indican days.	Duration of sugar days.	Result.	Notes.
1 N. S.	M	5¼	—10	+	+	+	..	..	.....	.....	Ac.	ac	1018	0	0	+	++	+	1 Cons.	6	—23 oz.	—10	?	Imp.	No acidosis. 9 mos.
2 J. V.	M	4½	—4	+	+	+	+	..	.....	.....	...	ac	1020	VST	0	++	+	0	n/	10	—20 oz.	—45	—28	Slow	Poor parental co-operation.
3 A. S.	F	12	—12	+	..	+	..	..	.....	+++	...	ac	1018	9	VST	+++	+++	++	n/	.....		..	..	Lost	Refused diet, etc.
4 M. W.	M	8	—7	+	+	+	..	+	++	.....	...	ac	1023	ST	0	++	++	?	n/	20	—96 oz.	—20	..	Imp.	
5 J. W.	M	6	+2	+	+	+	..	..	+	Petit mal	...	ac	1018	VST	0	++	++	++	n/		?	?	?	Cure	Petit mal.
6 L. W.	F	6½	—9	+	+	+	..	..	.....	.....	Vom.	ac	1024	ST	0	++	?	?	n/	20	—80 oz.	—14	?	Imp.	
7 G. W.	M	5	—10	+	+	+	..	..	.....	.....	...	ac	1018	0	0	++	+	?	n/	6	—30 oz.	?	?	Imp.	
8 J. H.	M	3	+2	0	+	+	+	..	.....	+++	...	ac	1019	VST	0	++	++	+++	Constip.	42	—144 oz.	?	?	Imp.	
9 B. C.	F	1¼	?16	+	++	++	?	..	+	.....	...	ac	?	VST	0	++	++	?	2–3	2	—18 oz.	14	14	Imp.	
10 D. B.	F	5½	—10	+	+	+	..	..	.....	.....	...	ac	1021	0	0	++	++	?	n/	2	—12 oz.	14	..	Imp.	
11 J. G.	F	6	?	+	+	+	..	..	+?	+?	...	ac	1026	VST	0	++	++	?	Constip.		?	21	21	Imp.	
12 J. K.	F	7	—1	..	..	++	..	..	.....	.....	...	alk	1016	0	0	+	+	?	?		?	21	no	Imp.	
13 C. R.	M	2⅛	—10	++	++	++	++	..	+	.....	...	ac	1030	ST	0	++	++	++	2 i. d.	3	—20 oz.	28	14	Imp.	
14 E. J.	F	2⅞	—30	++	++	++	+	..	+	Vom.	Vom.	ac	1021	0	0	+	++	++	Constip.		?	?	..	Imp.	T. B. Lungs as well.
15 D. H.	F	9	—11	++	+	+	..	..	.....	.....	Ac.	ac	?	VST	0	+++	++	++	n/	2	—32 oz.	25	20	Imp.	
16 C. B.	F	4¼	?	++	..	+	..	..	.....	.....	Ac.	ac	1024	VST	0	+	+	?	.....	5	—36 oz.	35+	35	Imp.	
17 M. B.	M	5½	12	+	..	+	..	..	+	.....	...	ac	1016	0	0	++	+	..	Constip.	5	—24 oz.	32	..	Imp.	
18 D. B.	M	6	?	+	+	+	..	..	.....	.....	...	ac	1023	ST	0	++	?	0	Constip.		?	?	?	Lost	13 mos. later splenectomy for Gaucher's disease.
19 R. F.	M	1.8	12	..	++	+	+	..	.....	+	...	ac	1024	0	0	+	+++	?	2 i. d.	5	—24 oz.	?	0	Imp.	
20 B. G.	M	4½	15	..	..	++	..	..	+	.....	...	ac	1024	VST	0	+	++	0	1 i. d.	5	—36 oz.	?	?	Imp.	
21 P. G.	M	7	3	..	..	+	..	..	.....	.....	...	ac	1021	0	0	++	++	0	1 i. d.	16	—38 oz.	?	0	Imp.	
22 A. C.	M	8	?	..	..	++	..	..	.....	.....	...	ac	1023	VST	0	++	++	?	Constip.	6	—64 oz.	—40	—20	Imp.	
23 J. C.	M	3	?	+	++	++	+	..	.....	.....	...	ac	?	0	0	++	++	++	3 i. d.	8	—78 oz.	?	?	Imp.	
24 K. C.	M	6	Normal	..	++	++	..	..	++ Lead colic	.....	...	ac	?	VST	0	+++	+++	++	Constip.	30	—60 oz.	—21	—14	Imp.	
25 J. C.	M	2.8	+2	..	..	++	..	+	+	.....	...	ac	1021	VST	0	++	++	+	n/	5	—26 oz.	—5	—5	Imp.	
26 G. C.	M	10	—11	+	+	+	+	+	+	.....	...	ac	1018	0	0	+	+	0	Constip.	7	—48 oz.	?	?	Imp.	
27 C. H.	M	7	?	+	..	+	..	..	+	.....	++	ac	?	VST	0	+	+	0	?	5	—26 oz.	?	?	Imp.	
28 J. A. C.	F	5	—16	..	..	+	..	..	+	.....	...	ac	1019	VST		+	+	?	n/	26	—48 oz.	?	?	Fair	
29 G. M.	M	5	—9	+	++	+	..	..	.....	.....	...	ac	1019	ST	0	+++	+	?+	?	5	—18 oz.	—7	—7	Poor	Failure to adhere to diet. Recurrences.
30 I. D.	M	6	4	..	++	+	..	..	+	.....	...	ac	1020	0	0	+	+	0	Constip.	30	—74 oz.	?	?	Imp.	
31 F. H.	M	3½	?	+	+	++	..	..	+	.....	+	ac	1023	VST	0	++	+	?	Constip.	20	—82 oz.	—5	—5	Imp.	
32 A. N.	F	1¾	—12	++	++	+	..	..	.....	.....	Vom.+	ac	1022	V SPT	0	+	+	+	2 i. d.	42	—160 oz.	—3	—3	Imp.	
33 E. O.	F	7	—14	++	+	++	..	..	.....	++	...	ac	1025	VST	0	++	+++	++	?	12	—80 oz.	—1	—1	Imp.	Recurrent convulsions.

+ = Plus. — = Minus. Ac. = Acidosis. ac. = Acid. Vom. = Vomiting. Imp. = Improved. n/ = Normal.

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CLINIC OF DR. JOSEPH I. GROVER

CHILDREN'S HOSPITAL

ENURESIS

February 2, 1920.

THIS boy was about as pronounced a case of enuresis as you will ever see. He came to this clinic first on June 10th of last year. He was subject to bed-wetting ever since his birth. He usually wet the bed two or three times every night and had never missed a night that the mother could remember. Besides that, he also had to pass his water very frequently all day long, and he wet his clothes three or four times a week. His mother thought that he could urinate every fifteen minutes. He was also occasionally incontinent of feces in the daytime, and had two such accidents during the month previous to the time that I first saw him.

His family history is negative except for the fact that his mother and her sister had enuresis when they were children up to about twelve years of age. This boy has a brother and a sister, but they are normal.

His past history tells us that the only disease he ever had was measles when he was three years old. He had his tonsils and adenoids removed when he was five years old, and he was circumcised when he was six years old. Both of these operations were done to help his enuresis.

On physical examination I found a normal temperature, and his weight was 47 pounds. He was just eight years old. He was a well-developed and well-nourished boy, apparently in good health. He looks a little better now than he did eight months ago. He has blonde hair and light blue eyes. His teeth showed two rotten stumps and three other small cavities. His throat was normal, the tonsils having been removed. His heart was normal except for a hemic murmur at the base. His lungs were clear

and the d'Espine sign was negative. The abdomen was negative. The reflexes were normal. His penis and scrotum were normal, and there was a good result from the circumcision.

The urine was amber and had a specific gravity of 1012. It was acid. There was no sugar or albumin. The sediment showed nothing of account.

His hemoglobin was 75 per cent. by the Tallqvist method.

When I first saw him I asked his mother several questions about his habits and mode of living, and I found that his appetite was poor. Here is an outline of his diet for the two days just before I first saw him.

June 8th.—Breakfast: Fried egg. Cup of cocoa with two teaspoonfuls of sugar.

Recess: Apple.

Dinner: Broth. Potato. Apple pie. Tea.

After school: Two pieces of cake and 3 cents' worth of candy.

Supper: Chicken. Potato. Cake. Cocoa.

June 9th.—Breakfast: Cocoa. (Would not eat anything else.)

Recess: Banana.

Dinner: Soup. Doughnut.

After school: Ice-cream.

Supper: Baked beans. Brown bread. Cocoa.

June 10th.—Breakfast: Cocoa. Nothing else.

He usually went to bed at about 9 to 9.30 P. M. He went to the moving-picture show every Saturday afternoon. In school he was fairly proficient in his work, but was nervous and irritable and couldn't sit quiet a minute. He was rather disobedient. He was naturally sensitive about wetting his clothes, and was made especially nervous and irritable by the taunts of his family and play-fellows. He slept very soundly, and his mother used to have quite a job to wake him up to pass his water.

He was naturally constipated, and when he was given a laxative he was often incontinent of feces the next day.

Before his mother brought him here for treatment she had tried a number of things to cure him, but without any success.

He was seen three years ago by their family doctor for his enuresis, and was advised to have his tonsils and adenoids removed. The operation was performed, but it had no effect upon his bed wetting, clothes wetting, or incontinence of feces. One year later the mother was advised by the same doctor to have the boy circumcised. The operation was performed successfully, but it had no appreciable effect on his condition.

Last year the boy was given a treatment with atropin. He was given increasing doses for a week, and this was repeated three or four weeks in succession, but no effect was noticed on the boy. One month before I first saw him an alkaline medicine was given and the urine rendered alkaline for ten days. This treatment likewise had no effect on the wetting.

The treatment that I outlined when I saw him first is for the most part contained on this printed sheet, which was given to the mother. Here is a copy of the outline:

Directions for Enuresis.—Give child as little liquid as possible after 4 P. M. This includes water, milk, and soup. He may have a wineglass of water with his supper.

Do not let the child run around after 4 P. M. It is very important to keep the child in a chair after 4 P. M.

Have the child sleep alone, and see that the windows are wide open.

Have the child go to bed at 7 P. M.—no later.

Give the child just three meals a day—no food at all between meals.

Do not let the child eat any candy, cake, ice-cream, soda, tonic, cookies, sweet or milk chocolate. Get along without any sugar on cereals if possible. Do not give jams, jellies, or syrup. No raw apples or bananas. No egg-nogs.

Do not let the child drink coffee, tea, or cocoa. Do not let the child have meat broths. No salty or highly seasoned foods, like pickles, mustard, catsup, salt or pickled fish, corned-beef, frankforts, baked beans, etc. Use as little salt on food as possible.

Do not let the child have meat or eggs for supper.

Pick up the child to pass water at *exactly* 10 P. M. and 6 A. M.

DIET LIST

Milk	Stale bread	Peas
Butter	Toast bread	String beans
Soft-boiled eggs	Milk-toast	Spinach
Scrambled eggs	Plain crackers	Asparagus
Dropped eggs	Barley cereal	Summer squash
Boiled fish	Oatmeal	Orange
Lamb chop	Pettijohn	Baked apple
Mutton chop	Cream of Wheat	Apple sauce
Roast chicken	Wheat germ	Stewed prunes
Boiled chicken	Farina	Junket
Roast lamb	Rice	Custard
Roast mutton	Hominy	Cornstarch pudding
Beef-steak	Tapioca	Bread pudding
Roast beef	Baked potato	Rice pudding
Plain macaroni	Mashed potato	Blanc mange

Besides the above directions, the mother was also advised to keep the child out of school for the remaining few weeks of the term. Moving-picture shows were also prohibited.

Then I outlined a schedule for the hours he was to empty his bladder. He was to urinate at 7 P. M. when he went to bed. Then he was to be waked up to urinate at exactly 10 P. M. and 6 A. M. During the day he was to be encouraged to empty his bladder once every half-hour—exactly on the hour and half-hour when the clock struck.

The mother was advised not to have the boy reprimanded for wetting the bed, but if he was caught breaking any of the treatment rules he was to be severely punished by spanking. His poor teeth were to be extracted or filled. No drugs or medication of any kind were ordered.

All of these directions were explained carefully to the mother in the presence of the child. I tried to make the reasons for each detail clear to her, and I took over half an hour to do it.

The mother remarked after the treatment was outlined to her that she realized it would be almost impossible to carry out the instructions, but she would do her best. When I asked her what part of the treatment she thought would be especially difficult, she said that to deprive him of baked beans would break his heart. But I made her promise that she would not have

baked beans in the house for two months, and I think she kept her promise.

I saw him the second time on June 18th. He weighed 46½ pounds. His appetite was poor because most of the things he was accustomed to eat were restricted, so he ate very little indeed. His bowels were still constipated, but he had had no incontinence of feces that week. He wet his clothes on only one occasion, and that was when his mother was out to the store, four days before. At night his bed was wet once by 10 o'clock and always wet by 6 o'clock in the morning. As far as could be ascertained he had broken none of the dietary or rest rules. The mother had taken him to a good dentist and his teeth were now in fine shape.

The only change I made was that he was to urinate every forty minutes during the day instead of every thirty minutes. I also advised his urinating at 2 A. M. in the middle of the night. The mother and boy were to be awakened by an alarm clock at that hour.

His third visit was June 24th. He weighed 46½ pounds. The boy entered the room looking very proud of himself. He had had no fecal incontinence. He had not wet his clothes once since the last visit. At night he was always dry when awakened at 10 o'clock; he was wet once at 2 A. M. and twice at 6 A. M. His appetite was better, as he was becoming reconciled to his new diet. He ate cereal and drank milk, which he had rarely done before. The only change I made was that he urinate every fifty minutes during the day instead of every forty minutes. No change was made in the night régime, *i. e.*, at 7 and 10 P. M., 2 A. M., and 6 A. M.

The fourth visit was on July 8th. He had had no bowel incontinence since the first visit. He had not wet his clothes once during the day. He was found wet once at 6 o'clock in the morning and had not wet the bed on any other occasion.

I had him urinate every hour during the day instead of every fifty minutes.

His fifth visit was on July 22d. He had not been wet day or night for sixteen days. I lengthened the interval from every

hour to one and one-quarter hours during the day. His sixth visit on August 11th showed his weight to be 47¼ pounds. He had had no "accidents" for a full month. The treatment outlined was that he should urinate every one and one-half hours during the day instead of every one and one-quarter hours, and no change was to be made from the four-hour schedule at night.

On his seventh visit, September 1st, his weight was 47½ pounds. There had been no wetting at all. His appetite was never better. He had been faithful to all directions. I told him to urinate every two hours during the day instead of every one and one-half hours, and to omit the 2 o'clock urination in the middle of the night.

On September 20th, his eighth visit, he weighed 47½ pounds. He could hold his urine for two hours during the day and went from 10 o'clock at night to 6 o'clock in the morning without difficulty.

The mother volunteered the information that the child was no longer nervous or irritable, and she wondered if he could again attend school. The boy seemed to have complete control over himself, and school was advised. I told him to be sure and urinate just before going into the school-room, again at recess time, and again upon dismissal. I advised the mother to continue putting him to bed at 7 o'clock and to pick him up at 10 P. M. and 6 A. M. as before.

His ninth visit was last October. His weight was 48¼ pounds. He had no further trouble. He was awakened a 10 P. M. to urinate, but got up himself at about 6 to 6.30 in the morning to go to the bath-room. I told him he might play a little after school. I didn't make any additions to his die list—contrary to his expectations. He was still to be awakened at 10 P. M. for urination.

Today is his tenth visit. He weighs 49½ pounds. He is probably cured of his enuresis. His mother says she is no longer punctual about getting him up at exactly 10 P. M., but calls him when she goes to bed. She asks me if he can now have candy and baked beans occasionally. I told her he could not.

The cause of the enuresis in this boy was evidently not due to adenoids or elongated prepuce or acid urine, as these factors had been eliminated previous to his present treatment. The etiology of enuresis is well exemplified in this case. I believe it is a general nerve-muscle fatigue due to mental strain, lack of sufficient sleep, excessive muscular exertion, and a poor diet. The average case is so utterly fatigued that at night it is almost impossible to wake him up to urinate; and often after an unsuccessful attempt the child is put back to bed and unconsciously empties the bladder five minutes later.

Most cases of enuresis, like this patient, begin the wetting in infancy. A few, however, begin later on, usually during the third or fourth year, after a normal period. The etiology of this latter variety is also a general neuromuscular fatigue, but the immediate cause is often found to be an acute disease like scarlet fever or chorea, or perhaps a fright or a fall.

The incontinence of feces has the same etiology as the incontinence of urine and is seen occasionally complicating the most pronounced cases of enuresis. After treatment is begun the incontinence of feces always clears up first, as it did in this boy.

The day wetting requires a little more explanation. Our patient had always had enuresis since birth. He was a day and night wetter combined. His bladder was never able to hold more than 2 or 3 ounces of urine at a time. When this capacity was reached the bladder had to empty itself. Normally the bladder stretches itself more and more as the child grows until it can hold more than 10 ounces. In this case the bladder remained contracted with more or less inelastic walls. In the treatment of cases like this one we must consider not only the general nerve-muscle fatigue but also the contracted and undilated bladder. By gradually increasing the interval of micturition during the day the bladder becomes accustomed to holding greater and greater quantities of urine, and stretches itself accordingly. This process requires weeks or months for success. It cannot be hurried, as the bladder stretches slowly.

Regularity of micturition is extremely important. The

bladder that has been used to emptying itself at "any old time" must be trained to regularity. Habit is an important factor in the etiology of enuresis. After the neuromuscular fatigue has been overcome the habit of unconsciously emptying the bladder may remain. This habit may require more time and patience to correct than the treatment of the nerve-muscle fatigue. The involuntary act of emptying the bladder must be made a voluntary one. He must be made to pass his water on the minute the same time every day. The interval must be short enough at first to keep him from wetting himself, even if the interval is as short as every ten minutes. The interval should not be lengthened more frequently than once a week, and preferably not more than five minutes at a time. I started this boy on a half-hour interval and increased it ten minutes at a time each week.

The day wetting usually clears before the bed wetting, because while he is awake he can consciously hold his water for a few minutes even if it takes a lot of effort. During the night, however, the whole act of micturition is involuntary, and he has no knowledge of what is taking place. He cannot help himself even if he were very desirous of doing so.

Every day and even every hour that the child is dry makes it easier to keep dry the next day or hour. The creation of the "dry habit" is very important for the final result. Frequent and regular intervals for urination tend to bring about the dry habit. The interval, however, must vary according to the severity of the case.

Children that wet the bed only and are normal during the day do not have contracted bladders, but weak ones—a part of the general nerve-muscle fatigue that I spoke of before. This general fatigue does not necessarily mean a thin, anemic child, but one with tired nerves and muscles due to lack of sufficient sleep, too much play and excitement, and improper food. Improper food tires the stomach and intestines, and, in turn, tires the whole system. So many factors take part in producing the general fatigue that unless every one is corrected the result will be poor. A careful diet will not end the wetting if the child

plays too long or goes to bed too late. A careful diet and plenty of sleep will not help matters if the regular periods of micturition are not adhered to strictly. If tuberculosis or some other wasting disease is present in the patient, the simple hygienic measures I have outlined will not, of course, correct the general fatigued condition, and tuberculosis must always be considered if the child does not pick up on the ordinary dietary and hygienic rules.

The diet is very important. Almost every case of enuresis that I have ever seen was found to be on more or less of a poor fare, and you will find it so if you take the trouble to investigate it properly. Sweets and sours must be omitted because they spoil the appetite and the proper digestion of the plain foods. Soups, broths, salty, and highly seasoned foods are either indigestible or diuretic. Coffee, tea, and cocoa are also diuretic. Raw apples and bananas interfere with the appetite and cause no end of indigestions. Eggs and meat are omitted for supper because they take too long to digest and interfere with restful sleep. No food is given between meals in order to rest the gastro-intestinal tract and, in turn, the whole system.

Besides the long hours for sleep there are other ways of resting the body. You must allow less exciting and boisterous play; prohibit the movies, music lessons, and school home lessons. A nap for an hour or even half an hour at noon may help a lot in the children that do not seem to get enough rest at night in spite of long hours of sleep. Sitting down after 4 P. M. affords a little more rest.

Bed wetting is normal in infants and small children, but becomes abnormal at about four years of age. Bed wetting as a family trait is very noticeable in reviewing the histories of a large series of cases. Enuresis is a common symptom in idiocy, spinal cord lesions, diabetes, and pyelitis, and when part of some other condition it cannot be treated with success as I treated this boy. Pin-worms may aggravate an enuresis because of the irritation about the anus. If they are present they should be treated first before anything else is begun.

Blonde children are more subject to enuresis than brunettes,

especially the very light blondes with light blue eyes. Girls and boys are equally affected.

Nervousness usually accompanies enuresis. Habit spasms and stuttering are common accompanying conditions. As the child recovers from the enuresis during treatment, it is surprising how the nervousness and irritability are benefited. The nervousness and habit spasms have nothing to do with the enuresis, but have the same etiology. So closely related are nervousness and enuresis that enuresis has often been called "bladder stuttering."

After a patient has been apparently cured it must be seen to that he remains permanently cured. This means a continuance of the strict régime for months afterward until the bladder can be surely trusted. Most mothers cannot usually see this, and begin to break rules a week or two after the child has had its last "accident." Some slight change is made in the child's routine and he starts wetting all over again.

It has been my experience in treating enuresis in the way I have described that about 20 per cent. are cured within the first day or two, and never wet again after that. About 20 per cent. more are cured after three or four weeks, and all but about 1 in 10 or 12 are either cured or greatly relieved. By "relieved" I mean that they wet no more than once a week. The incurables are usually found to be either tuberculous or slightly feeble-minded. Once in a while you will find one with an intermittent bacteriuria. The poorest results are encountered in children who live with some relative other than the mother or with a stranger who boards the child. These guardians invariably believe that the wetting is the child's own fault and rarely believe enough in the soundness of the treatment to give it a fair trial. I always give these guardians a poor prognosis, and tell them why.

As a great deal or, in fact, all of the success of the treatment lies in following the directions, the attitude of the child must be considered. Many little children four or five years old do not care whether they wet the bed or not. These children that do not appreciate their condition or feel no shame will not follow

directions. They will not sit quietly when told and will steal water and eat candy at every opportunity. They also cry and fight to stay up late at night, so much so that they often get the best of their "easy" parents, and the enuresis fight is lost. The older the child and the more mature it is, the easier it is to cure it, because of the co-operation in following directions. It was because this boy was anxious to get better that he followed directions, and he says he is glad of it now. I am going to see to it that he sticks to the simple life and diet so that he doesn't begin his old tricks again.

CLINIC OF DR. PHILIP H. SYLVESTER

CHILDREN'S HOSPITAL

A CASE FOR DIAGNOSIS

THE patient, J. G., 3d, was the only child of young and healthy parents. He was born normally at full term and weighed 7 pounds and 4 ounces. He was fed wholly on breast milk until eight months old. During and following weaning his diet was regulated by good pediatricians. At one year he weighed 22 pounds. He has had a few colds, but up to one year had never been really sick. His father has been in the army and his parents have moved about a great deal, so that he has been under the care of a great many doctors. In spite of a very sound diet he had not gained very much during the last several months and has had several attacks of what has been called "acidosis." These attacks usually come two or three weeks apart and last from one and a half to three days. He is now eighteen months old and for the last several months has had the following diet:

Whole milk	1 quart
Eggs	1
Meat (chopped)	1 to 2 tablespoons
Cooked cereals	6 to 10 tablespoons
Strained vegetables (green)	2 tablespoons
Potato (small, baked)	1
Fruits (orange juice, prune juice, apple sauce)	
Spinach	1 tablespoon twice weekly

He is usually pretty lively, feels well and happy, and has a good appetite. His bowels move once daily without aid. He has never had any urinary symptoms. Two weeks ago he weighed 23 pounds and 2 ounces, representing a total gain of only 1 pound and 2 ounces in six months.

He has been sick with "acidosis" for four days. This attack has been similar to, but more severe, and has lasted longer than the usual previous attacks. The mother has carried out the previously advised treatment which consists of starvation, and 15 grains of bicarbonate of soda every two hours, with only "just enough water to wash soda down." After two days of this treatment the baby seemed somewhat better and whole milk was given in undetermined amounts. Yesterday (third day of attack) the child vomited its milk twice and today twice. There has been a little fever, not taken by thermometer until today. He has been very fussy and has seemed quite uncomfortable, but has not cried as if in pain. The bowels have moved daily with cathartics and for the last two days the stools have been almost white. There have been no urinary symptoms. Tonight the child has refused soda, milk, and water, and has a temperature of 102° F.

Physical examination shows a well-developed and nourished child of about fourteen months in apparent age, moderately prostrated, whining and fussing, with a grayish pallor. The anterior fontanel is closed; there are 14 clean, strong teeth, none erupting. The throat is very red, with pin-points of grayish exudate on the tonsils. The ears are negative. There are no glands of the neck; heart and lungs fail to show abnormalities; the abdomen is flat, a little tender in the right upper quadrant, where the liver edge is found to be 1½ cm. below the costal border. The spleen is not felt. No masses, other than the liver, are felt. The skeleton shows no evidence of rachitis. Genitals and extremities show no abnormalities. Nervous system: pupils react equally; no retraction or stiffness of neck; knee-jerks equal; no Kernig or Babinski. Stool passed during examination is white with a rather cheesey odor.

Diagnosis, Differential.—1. *Acidosis.*—From the mother's story of several similar but less severe attacks, the diagnosis of "acidosis" is refreshingly appropriate, but I have long since learned to accept with reservations any diagnosis of "acidosis" unaccompanied by the respiratory signs. In other words, when a child has enough "acidosis" clinically to amount to anything

he is drunk and shows it. Again, acidosis is seldom if every primary, but usually is a symptom of a readily ascertainable primary condition. Ordinarily we see five types of conditions which are called acidosis:

A. Acidosis characterized by vomiting and *fever*. These attacks are more or less periodic and are often called cyclic or recurrent vomiting. Studied cases always have a leukocytosis as well as fever, and almost always have an obvious focus of infection, such as tonsillitis, otitis media, appendicitis, etc.

B. Periodic, cyclic, or recurrent vomiting. These cases have periodic attacks of vomiting and do not retain even water. Severe cases have hyperpnea, acid bodies in the urine, diminished CO_2 tension, and act very drunk. Careful study will almost invariably reveal a long-standing digestive disturbance, usually associated with the indigestion or overfeeding of fats (Kerley's suboxidation). They seldom have fever or leukocytosis.

C. True acidosis with *diarrhea*. These cases are always quite severe, and have, without question, acidosis, but the acidosis is relative and due to loss of not only salts but *water*.

D. True acidosis with sudden death in hours or days. Cases coming to autopsy invariably show lesions of the central nervous system indicating infection, usually encephalitis. These often resemble the "pink lesions" of infantile paralysis.

E. Urinary or symptomatic acidosis. In practically every case accompanied by fever acid bodies will be found in the urine. Unless there is severe vomiting or hyperpnea these findings may be absolutely disregarded.

Treatment of Acidosis.—Biochemical research and clinical experience gathered during the last several years have shown without question that acidosis is the result of abnormal combustion of the body tissue fat and protein. Just what determines the combustion of these elements is unknown. Infection undoubtedly plays a part, fat overfeeding and chronic digestive disturbances likewise.

Essentially, then, if the tissues—fat and protein—have started to burn, and if the by-products of their combustion are the poisonous acid bodies, it would seem, if a soluble fuel *more*

readily available for combustion were provided, that this soluble fuel might be burned without poisonous by-products and the tissues left untouched. Therefore, it is reasonable to assume that sugar, than which there is no more soluble or available carbohydrate, may be given with benefit. This may well be done by means of orange-juice, which sweetened with honey, glucose, or even cane-sugar, has been found to take care of, in hours, any acidosis except Types C and D.

For Type C it must be remembered that the acidosis is due to the loss of salts by the bowel, and therefore soda will be indicated. It must, however, be emphasized that the gravity of the child's condition is due more to the loss of water than salts, thus rendering the blood so concentrated that the kidneys can do very little of their work, so that to add soda or salts to the already great burden will only increase the difficulty. The sensible treatment is to replace the lost fluids by any means possible; by mouth if the child will swallow, by the longitudinal sinus, intravenously, and subcutaneously. Rectal infusions are seldom retained and often do more harm than good.

For Type D the acidosis is secondary and entirely symptomatic and, of course, nothing can be done. Intelligent parents usually request an autopsy, the findings of which generally satisfy them that death was unavoidable.

2. *Infectious Jaundice.*—The season of the year, the presence of many other cases, the enlarged and tender liver, and the white stool make this diagnosis reasonably probable. Jaundice of the sclera was not noticed, but the light was artificial.

3. *Infection of Throat.*—There is no doubt that the throat is infected. It is well known that throat infections frequently cause digestive disturbances, and that a large proportion of cases seen for acute indigestion show on examination an acutely infected throat.

4. *Recurrent Vomiting.*—Based on the story, this diagnosis seems very reasonable, but the diet has been unusually good and not high in fat. It must be admitted, however, that the white stools certainly suggest fat indigestion.

5. *Protein Indigestion with Intoxication.*—Many other chil-

dren in this community had at this time similar attacks with moderate vomiting, fever, leukocytosis, acutely infected throats, and white stools. These stools invariably showed no excess fat, but large amounts of undigested protein (mostly casein). These children did not in any way resemble, clinically, the cases rarely seen of acute protein indigestion with putrefaction, characterized by tremendous prostration, subnormal temperature, and foul, brown, musty, alkaline diarrhea. They were simply quite a little knocked out, vomited, and had large cheesey stools.

Laboratory Findings.—White count, 24,000. Urine, concentrated, acid, 1024, albumin 0, sugar 0, acetone faint, diacetic 0, bile 0. Stool white, alkaline, moderate soaps, protein 4 plus (casein).

Final Diagnosis.—Acidosis is ruled out because of lack of clinical and urinary evidence.

Infectious jaundice is ruled out owing to lack of bile in the urine and failure to find jaundice by daylight.

Recurrent vomiting (fat intoxication) may be ruled out on the ground that for a long time the diet has been very good and that stool examination shows no excess fats. In addition, the urine is negative for acid bodies.

Infection with acidosis may be ruled out on the ground that while infection undoubtedly exists, as shown by the appearance of the throat, fever, and leukocytosis, yet there is no clinical evidence of acidosis and the urine is negative.

Infection with protein indigestion therefore remains and is the only diagnosis possible consistent with the clinical picture and the laboratory findings.

Final Diagnosis.—Acute infection of throat with secondary vomiting and protein indigestion (not putrefaction).

Treatment.—The throat infection will undoubtedly take care of itself in a few days, even if left alone. It is probable that every physician has a favorite treatment for throat infection which differs more or less from that of other physicians. My personal preference in treatment is based upon the theory that chlorate of potash is excreted by the lymphoid structures of the throat and therefore increases the secretions from these structures. In this

way infecting organisms are washed away from the tonsils, swallowed and destroyed, and absorption is lessened. The child was, therefore, given chlorate of potash in solution, 2 grains every two hours. Inasmuch as the adenoids and the entrance to the eustachian tubes are in babyhood close neighbors to the tonsils, and therefore extremely susceptible to infection by direct extension, it is wise to try to prevent that extension. This was done by means of a 10 per cent. solution of argyrol, 1 drop in each nostril every two hours. (Inasmuch as water will not run up hill it is necessary to turn the child upside down when administering any medication intended for the adenoids or eustachian tubes.)

The protein indigestion is probably not very important, yet it would seem wiser and safer to forestall the more serious protein putrefaction by withholding protein altogether. In view of the fact that in almost every case of acute digestive disturbance fat is poorly digested it would be wise to withdraw the fat also. Accordingly, the child was given nothing to eat but cereal jellies and strained orange juice. He seemed quite eager for and took large amounts of these. At the end of twenty-four hours his temperature was normal. At the end of forty-eight hours he felt and acted perfectly well and the movements were brown. He was then given his previous diet in full and did perfectly well.

Subsequent History.—During the following two weeks the child seemed as well as usual, at the end of which period he became suddenly sick again, was quite feverish, cried a lot, and vomited everything taken for the first twelve hours. The mother starved him and gave large amounts of soda, but the vomiting persisted. On physical examination there was an evident mild, clinical acidosis, the throat was red, there was gray exudate in the follicles of the tonsils, the white count was 25,000, and the urine showed large amounts of acetone and diacetic acid. These findings leave no room whatever for argument in making the diagnosis of acute infection of the throat with clinical acidosis. The same throat treatment previously described was given. The acidosis was severe enough to warrant treatment as such.

Inasmuch as acid bodies in the blood are very readily eliminated and completely so in a very short time, it seems more sensible to prevent their further formation than to try to neutralize them by soda, which bothers the child, tastes bad, and often tends to increase the vomiting. It has been my custom for several years to give strained orange juice or the juice of canned pineapple instead. These are taken eagerly and gratefully, even by very small babies. My rule is to give either, *ice cold*, in teaspoonful doses at five-minute intervals. When there has been no vomiting for half an hour the dose is doubled, and that again doubled at the end of each half-hour when there has been no vomiting, until 1 ounce is retained, after which 2 ounces are given every two hours. Usually, even in reasonably severe cases, vomiting ceases altogether in two hours. This treatment was carried out, vomiting ceased shortly (I have no record of exactly how long it persisted), and the child was completely well in forty-eight hours.

For the succeeding three weeks the patient was vaguely unwell at times. He was very peevish, cried a great deal, ate poorly, and did not gain. Repeated examinations failed to show fever, digestive or urinary disturbances. The tonsils, however, remained rather red, had a granular appearance, and were obviously adherent to the pillars. The ear drums showed no abnormalities.

At the end of the above interval the child had another typical "acidosis attack" with fever, aciduria, and leukocytosis. The throat showed evidence of acute infection and the child was extremely irritable and peevish, and refused almost all food. After twenty-four hours of treatment of the throat and acidosis as outlined above the vomiting ceased and the throat appeared to be much better, but the child continued to be sick. Physical examination revealed nothing abnormal. Ear examination revealed slight blurring of the light reflex of the right ear, but no redness, and no tenderness in tube, canal, or mastoid areas. There was no edema of the posterior wall of the canal. During the next four days the temperature rose from 100° F. in the mornings to 103° and 106° F. at noon time, and dropped to

100° F. again in the evening. Repeated examinations revealed nothing further than the symptoms above described. The white count was 22,000, 23,000, and 25,000. Conditions considered were:

1. General infection arising from tonsillar infection.
2. Otitis media.
3. Eustachian tube infection without signs in drums.
4. Meningitis.
5. Lateral sinus thrombosis.
6. Mastoiditis.
7. Central pneumonia.
8. General tuberculosis, lighted up by previous infection.

Discussion.—(1) In any general infection in infancy or early childhood the child invariably looks badly and seems very sick. Furthermore, the heart is liable to dilate rapidly and to develop murmurs. Also, with diminishing resistance, the leukocytosis is liable to decrease. In this case the child looked and acted surprisingly well. The heart did not dilate or develop murmurs and the leukocytosis increased a little.

(2) Beyond a slight blurring of the light reflex of the drum there was no evidence, whatever, of any trouble in the ears.

(3) It is well known that infection can exist within the eustachian tube for several days before any evidence of it appears in the ear drums. Owing to ready absorption these cases appear acutely sick and are very puzzling on account of the lack of any definite signs. They usually, however, are quite tender to pressure in the so-called tube area, that is, in the apex of the triangle formed anteriorly by the ascending ramus of the jaw and posteriorly by the anterior edge of the mastoid. This child showed no such tenderness.

(4) Early meningitis is to be considered even in the absence of any definite nervous symptoms. The child was very irritable and cranky, and while it wanted to be held all the time, it did not want to be handled, and seemed unduly sensitive.

(5) Sinus thrombosis should be considered in all cases having a high and irregular temperature and a negative physical examination, with negative urine. Such cases are, however, ex-

tremely prostrated, look very sick, and often fall or nod as if dizzy.

(6) Mastoiditis doubtless may exist without any of the usual external symptoms whatever. Among older children tenderness and edema over the mastoid process are classical, but in babies edema of the posterior wall of the canal is frequently the only sign.

(7) A central pneumonia or other deep infection of the lungs may exist for days without any evidence on physical examination. As a rule the respirations are characteristic enough to arouse suspicion at least, if not to justify the diagnosis.

(8) General tuberculosis is often enough stirred up by other processes to justify the suspicion that it might exist here.

At this point roentgenograms were taken of the lungs and the mastoid and Dr. John Lovett Morse was called in consultation. Physical examination by him revealed nothing new. On considering the evidence to date it was agreed that:

(*a*) The child was not sick enough to warrant a diagnosis of general infection.

(*b*) There was no evidence whatever of otitis media.

(*c*) There were not enough disturbances of the nervous system to justify lumbar puncture in order to rule out meningitis.

(*d*) Roentgenograms of the head showed no occlusion of mastoid cells, and therefore ruled out mastoiditis.

(*e*) Roentgeongrams of the lungs showed no abnormal shadows and therefore ruled out central pneumonia.

(*f*) While the negative tuberculin skin reaction is of no value in a case of general tuberculosis, it is inconceivable that so acute a case, if tuberculous, should fail to show mottling in the roentgenogram of the lungs.

It was also agreed that, while we could find nothing suggesting otitis media, yet the opinion of a competent aurist would be of value in ruling out any infection of the accessory sinuses, eustachian tubes, or lateral sinuses, or, perhaps, in pinning down the infection to somewhere above the neck, as we believed that all

other processes had been ruled out. Dr. E. A. Crockett was then called in consultation and, after examination, decided that:

1. Roentgenograms ruled out mastoiditis.

2. The child was not sick enough for, and the absence of inequality of the external jugulars ruled out, lateral sinus thrombosis.

3. On account of slight tenderness in the tubal areas a low grade infection existed in the eustachian tubes which had not yet revealed itself in the appearance of the ear drums, but which probably would be definite within two or three days.

4. The appearance of the tonsils and the course of the infection in the eustachian tubes was characteristic of chronic infection which was primary in the tonsils and adenoids, and should warrant their removal as soon as the present infection had died down.

Immediately Subsequent History.—On the following morning it was reported that the child had spent a very uncomfortable night, had cried a lot, and had repeatedly put his hand to his right ear. Examination showed a red and yellow bulging ear drum. Paracentesis was followed by a few drops of thin pus and dark blood. Examination of the left ear showed a slight blurring of the light reflex, but no redness or thickening. The tube area, however, was quite tender. That afternoon the temperature rose again to 105° F. The following morning the left ear drum was found to be reddish yellow and bulging and paracentesis was followed by a gush of blood and pus. The temperature did not rise again, the ears discharged profusely for four days, and then gradually dried up; and the child seemed perfectly well at the end of ten days.

Final Diagnosis.—1. Chronic infection and hypertrophy of adenoid, encroaching on fossæ of Rosenmüller.

2. Chronic tonsillitis with hypertrophy and adhesions.

3. Subacute otitis media.

Final Treatment.—During all this period of six weeks or more treatment has been necessarily directed at secondary rather than at primary manifestations. There can be no doubt, in view of the development of these manifestations, that the tonsils and

adenoids are the seat of the primary infection and therefore to blame for everything. Treatment would be criminally incomplete and the child left with an assurance of continued trouble without their removal. It is generally agreed that the best time to remove tonsils is when the immunity from the last infection is at its height. That is from ten days to two weeks after the last infection has died down. When the ears have been infected. however, it is agreed that the best time to remove the tonsils and adenoids is when all evidence of infection in the ears have subsided. The ears were perfectly normal in three weeks, when Dr. Crockett performed the operation.

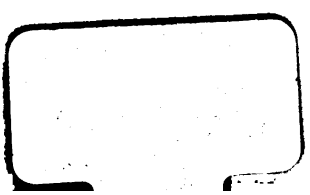
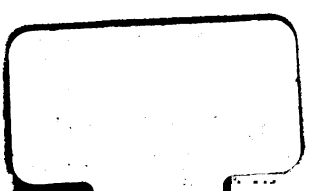
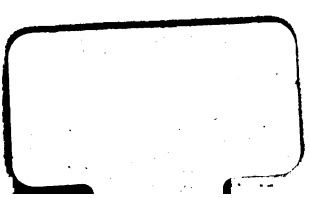
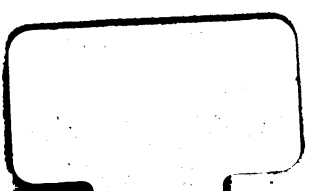
Subsequent History.—For the following three months the child seemed perfectly well, was very happy and lively, and ate tremendously. He gained 5 pounds. At the end of that time he had a slight sickness lasting for two days, was a little feverish, was cranky, and had a rash like "chickenpox." There was no vomiting or symptom of "acidosis." Examination revealed a slightly red throat and characteristic insect bites on the legs and back. Diagnosis: Pharyngitis and insect bites.

Conclusions.—It seems to me that several lessons are to be drawn from this case:

1. The futility of accepting a preformed diagnosis of "acidosis" from the history, and of considering such condition primary.

2. Infections of the eustachian tubes may exist for days without presenting evidence justifying even a shred of suspicion,

3. Even in babyhood not remarkably diseased tonsils may do a great deal of damage before they can be recognized as a source of other processes.

4. In certain cases prolonged study is necessary in order to differentiate each symptom or set of symptoms and to correlate such with the primary process.

5. To discover the primary focus is not enough. It must be removed before irreparable damage has been done to or through other structures.